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XII. TWO LIVERPOOL SURGEONS

II. EDWARD ALANSON, A PIONEER IN HOSPITAL SANITATION

EDWARD ALANSON, Surgeon to the Liverpool Infirmary, was far in advance of his time. Overcrowded hospitals, badly situated, and with little or no sanitation, had not yet attained the dreadful notoriety to which they were destined. Liverpool Infirmary perhaps would in any case have escaped the appalling mortality of some of the other general hospitals, for it was near the sea and the medical students were few. Nevertheless, Alanson foresaw the dangers, and made a series of remarkable recommendations, which he published in his *Practical Observations on Amputation and the After-Treatment* in 1779. He says :—

" The air in which the cure is to be conducted is a point worthy of your greatest attention ; if possible the room should be spacious and in an open wholesome situation. It is well known that in hospitals which are situated in populous towns and much crowded the salutary influence of the air is so altered that compound fractures and other important surgical cases prove peculiarly fatal ; and that amputations done in the country will be followed almost certainly with a speedy cure. The consequent symptoms are trifling, nearly the whole internal surface of the wound unites by the first intention ; the suppuration consequently is small and as soon as laudable, the secondary union takes place and the whole cure is speedily completed.

" Many hospitals are so tainted by unwholesome effluvia that they are rather a pest than a relief to the objects they contain.

" The following regulations are humbly recommended to the consideration of those who have the care of hospitals in want of such attention.

" 1. No ward should be inhabited for more than the space of four months together ; for it is impossible to keep a room healthy, that is constantly crowded with diseased people ; the walls should be scraped, whitewashed and every other necessary means used for the purification of the air before the readmission of patients.

" 2. The bed-stocks should be made of iron to prevent the lodgement of vermin and the more easy absorption of putrid matter.

"3. The bedding should be more frequently changed than is usually done; and the bed-tick stuffed with chaff, hay, cut straw, or materials of such easy expence as to admit of their being frequently changed.

"4. Where a hospital is conveniently situated for the purpose all the patients that are able should carry out their bedding and expose it in the open air for several hours every day when the weather will permit.

"5. On the days of admission those patients that have inhabited foul ships, jails, cellars or garrets, workhouses or other infected places; or whose clothes are dirty or suspected to contain vermin, before they are suffered to appear in the ward, should first be stripped and washed in the warm bath, and afterwards clothed with proper dresses, provided at the expence of the charity; by which means the evil of importing infection, so detrimental to the salubrity of every hospital, would be greatly remedied.

"6. The dresses for the men may consist, chiefly of a clean shirt, jacket and trousers; for the women, a shift, petticoat and bed-gown; the rest may be supplied from their own clothing, which will easily admit of being first well cleaned.

"7. The infected clothes should be baked in an oven constructed for the purpose; by which all vermin and infection will be destroyed; and the clothes may be returned clean to the patients, when they are discharged the hospital.

"8. The patients, when received on the days of admission, should be placed in the wards which have been last ventilated and not in those that have been long inhabited; where, it may reasonably be presumed, the air is considerably tainted.

"9. All incurable or infectious cases should be refused admittance; and amongst these should be classed old chronic ulcers of the legs, and particularly those in which there is a great loss of substance, for these seldom remain long healed; hence most hospitals are so crowded, that the intention of the charity is perverted, as the air is rendered unwholesome.

"10. All offensive, gangrenous, or other putrid sores, should be placed in distinct rooms provided for that purpose; and not suffered to taint a whole ward.

"11. There should be particular rooms provided for those patients who are the subjects of operations; they should be in the most airy situation, never long inhabited and alternately cleaned and ventilated, as before advised.

"12. A hospital should never be crowded on any account; and always of so large a construction that some part of the building may at all times be uninhabited for the purpose of white-washing, ventilation, &c.

"13. When any person has been affected with a putrid disease, or confined to bed for a length of time, let the bed be emptied and the bed-stocks, the bed, the sheets, and other linen be washed, and the rest of the bedclothes exposed for some time in the open air and baked in the oven, before they be used again.

"14. Let the nurses see that every patient's hands and face are washed every morning; and their feet, once a week.

"15. Let the nurse of each ward be liable to a fine, to be deducted from her wages, if some of the windows in her ward are not kept open during a stated number of hours every day.

"16. To every Infirmary, particularly where the wards are crowded, a house in the country, well situated, and at a convenient distance should appertain; without such assistance many of the patients must perish, which would be easily and certainly preserved; and it will be found the best policy in the trustees of an Infirmary

to provide such an appendix. By such assistance patients may be easily cured at a small expence; but, if suffered to remain in an Infirmary, their recovery will

EDWARD ALANSON

either be prevented, or obtained in a great length of time at a considerable expence by the most costly drugs, nutritious diet, &c. Likewise the house will be constantly crowded with the most miserable objects, to the anxiety of those who attend them

and the exclusion of other patients, who might have been cured in the interim. Many hospital-surgeons are under the necessity of providing lodgings in the country, at their own expence, for their patients who have undergone operations; rather than suffer the pain of a disappointment in compleating a good cure, or seeing their patient languish under a hectic, incurable in a crowded Infirmary. Therefore it is hoped that these considerations will influence the humane trustees, to provide these conveniencies for the poor sufferers."

It is evident that Mr. Edward Alanson, Surgeon to the Liverpool Infirmary in 1782, was almost exactly a century in advance of his time. There is no evidence that his suggestions were adopted at Liverpool or in any other hospital at home or abroad. There is evidence that he was a thoroughly good practical surgeon. He advocated amputation by a skin and muscle flap with circular amputation of the deep muscles when the majority of his contemporaries were content to remove a limb by the simple circular method. He tried to get union by first intention instead of by suppuration. He freed the arteries before he tied them when it was the custom to ligature nerves, arteries and the surrounding tissue in a single bunch, and he undertook with success such large operations as removal of the whole arm with the scapula. The first edition of his *Practical Observations* was a pamphlet of 64 pages, published in 1779; the second edition, enlarged to 296 pages, was published in 1782, and was translated into French by M. Lassus in 1784.

Alanson was born on Oct. 23, 1747, at Newton-le-Willows, Lancashire, the son of John and Margaret Alanson. His father was a large farmer and a man of substance. The son was apprenticed in 1763 to William Pickering, one of the Surgeons to the Liverpool Infirmary. He went to London in 1768, where he and Edward Jenner were amongst the first house pupils of John Hunter, who had just been elected Surgeon to St. George's Hospital. The fruits of his association with the Hunterian school are still to be seen in the Museum of the Liverpool University, where there is the body of a child with the label, " Injected and Dissected by Edw$^{d.}$ Alanson in Docter W$^{m.}$ Hunter's Rooms London March 1769."

Alanson returned to Liverpool in 1770, was elected Surgeon to the Infirmary, and in 1778 became one of the first three Surgeons at the Liverpool Dispensary. He lived with his former master, William Pickering, until in June, 1774, he married Miss Holland, daughter of a merchant. He joined practice with Henry Park (*vide* p. 205) in 1777, moved to Wavertree in 1790, resigned from the Infirmary in 1794 on the ground of ill health, retired to Aughton near Ormskirk, returned to Wavertree in 1808, and died there on Dec. 12, 1823, survived by his wife and five of his twelve children. It is noteworthy that neither he nor Henry Park ever became *Members of the Corporation of Surgeons*, though both styled themselves Surgeons.

I am indebted to Mr. R. W. Murray, F.R.C.S., late Honorary Surgeon to the David Lewis Northern Hospital and Liverpool Infirmary for Children, for information and for the portrait. He wrote in 1914 *Edward Alanson and his Times.*

ROTATION AT THE SHOULDER
A CRITICAL INQUIRY

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[This paper was completed but for the drawings in April, 1936. By the last mail (June, 1936) I received Codman's[1] monumental work on the shoulder. To the best of my knowledge this is the first copy of the book to reach Johannesburg. I find that Codman has preceded me in the discovery of certain facts which I had thought described for the first time in this paper. This is not to be wondered at in so acute an observer as his work demonstrates this great American surgeon to be, and I gladly acknowledge the precedence I indicate above.

In his book Codman makes two statements to which in particular my remarks refer: (1) On p. 43—"you can prove that the completely elevated arm is in either extreme external rotation or in extreme internal rotation"; (2) On p. 44—"The range of rotation of the humerus diminishes as it is elevated".

As this knowledge is of practical value I submit my communication unchanged.]

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I. INTRODUCTION

The humero-scapular joint is distinguished from all other joints of the body by its extensive range of movement, by the complexity of these movements, and by the fact, unique in human anatomy, that it is overhung by an osseo-fibrous arch which exercises highly important limitations on certain of the excursions at the joint. So many factors require analysis in an understanding of the mechanics at the shoulder-joint that there are still aspects of the subject regarding which knowledge is incomplete. Furthermore, Ridlon[2] expressed the general opinion of surgeons anent shoulder injuries when he said "the difficulties encountered in the treatment of injuries to the shoulder-joint have always been more troublesome and more obscure than the difficulties in treating all other joints put together"; and Lovett[3] remarked "that no common injury is so badly treated, save possibly the spine, as injuries to the shoulder". It is patent then, that any light on the manifold obscurities and perplexities of this inadequately understood joint may be of value in smoothing out some of these difficulties.

The objects aimed at in this paper are: (1) To indicate that our knowledge of rotation at the shoulder is incomplete; (2) To amplify this knowledge; (3) To make certain important surgical deductions from this fuller understanding.

II. REVIEW OF THE LITERATURE ON THE ROTARY PHASE OF SHOULDER-JOINT MOVEMENTS

Rotation of the head of the humerus receives but little space in the large modern text-books of anatomy, as the following extracts show.

Text-book of pure anatomy[4]: "The humerus is rotated outwards by the infraspinatus and teres minor; and it is rotated inwards by the subscapularis, latissimus dorsi, teres major, pectoralis major, and the anterior fibres of the deltoid".

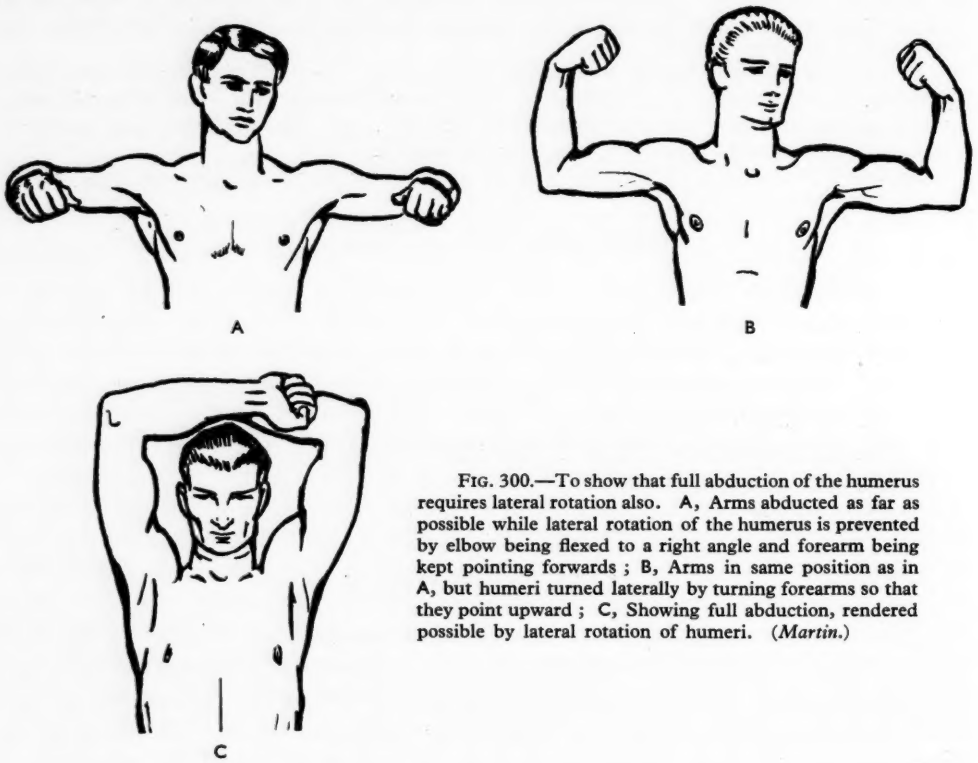

FIG. 300.—To show that full abduction of the humerus requires lateral rotation also. A, Arms abducted as far as possible while lateral rotation of the humerus is prevented by elbow being flexed to a right angle and forearm being kept pointing forwards; B, Arms in same position as in A, but humeri turned laterally by turning forearms so that they point upward; C, Showing full abduction, rendered possible by lateral rotation of humeri. (*Martin.*)

Text-book of pure anatomy[5]: Rotation of the humerus takes place "round a vertical axis drawn through the extremities of the humerus from the centre of the head to the inner condyle; in rotation forward (that is medial-ward) the head of the bone rolls back in the socket as the great tuberosity and shaft of the humerus are turned backward, i.e., lateral-ward". The muscles concerned in the actions are then indicated.

Text-book of surgical anatomy[6]: "Rotation of the humeral head normally approximates 180 degrees".

Text-book of orthopædic surgery[7]: "Rotation of the shoulder is greatest when the arm is partially abducted, and in a dissected joint the arc may approximate 135 degrees. At a right angle with the body under these circumstances it is about 90 degrees."

All these excerpts are taken from text-books which because of their great general worth are used almost universally. It will be seen that rotation at the shoulder-joint is not extensively commented on.

In 1932 Martin,[8] University anatomist at Trinity College, Dublin, made a contribution to the existing knowledge of rotation of the humerus which is quite the most important yet written. Surgeons taught for generations that most

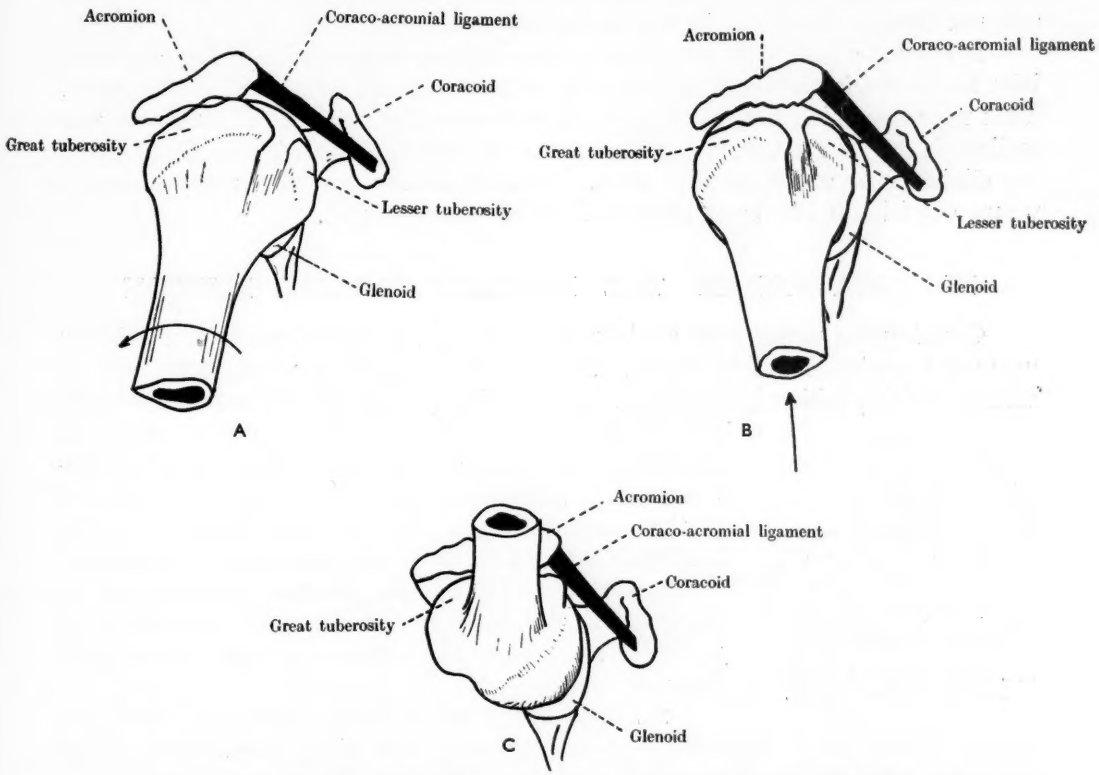

FIG. 301.—Demonstrating the part played by the osseo-fibrous arch overhanging the head of the humerus in the mechanism of abduction. A, The great tuberosity of the humerus is seen to impinge on the acromion; B, The humerus is rotated out; C, Abduction can now continue, as the tuberosity has receded beneath the acromion.

shoulder-joint injuries should be treated in abduction and full external rotation of the humerus, but it remained for an anatomist to show the scientific reason for this position of the bone.

The essence of Martin's paper is that, whereas abduction of the humerus to a right angle can be carried out whether the bone is rotated in or out, the second 90 degrees of abduction cannot be effected unless the humerus is fully rotated outwards. This is well shown in his lucid illustrations (*Fig.* 300, A, B, C). In *Fig.* 300, A the humerus is rotated inwards, being held in this position by the forearms being flexed to right angles and directed forwards. Very little further

abduction is possible. If the test is carried out on one side only, the result may be vitiated by tilting of the body. In *Fig.* 300, B the humeri are rotated out to the full, the forearms being flexed as before and directed vertically upwards. Now abduction can be carried to the vertical with ease (*Fig.* 300, C). Martin points out that during the second 90 degrees of abduction the great tuberosity of the humerus comes in contact with the lateral edge and under surface of the acromion and coraco-acromial ligament, and further abduction can only occur if the tuberosity slides under the acromion, which it can only do by passing backwards—i.e., lateral rotation of the humerus (*Fig.* 301).

The very important fact is adequately stressed that deficient abduction at the shoulder may be dependent on inability to effect lateral rotation of the humerus and not on any defect in the mechanism of abduction itself.

It has for a long time been held a wise precept that injuries of the shoulder-joint be treated in right-angled abduction and full external rotation of the humerus. That the explanation has followed many years after the principle of treatment was realized is due to the value of the latter having been appreciated, empirically, from the clinical observation of such cases. Surgical history can show many instances where empiricism has been justified later by scientific fact.

III. ROTATION OF THE HUMERUS DURING FLEXION

Carrying the upper limb straight forwards (in the sagittal plane of the body) up to the vertical is called flexion. Martin pointed out that whether the arm be carried to this position by flexion or by abduction (movement in the frontal plane), the position of the humerus at the end of the act is identical, i.e., the lateral condyle points straight backwards. The inference is that the mechanism of rotation of the humerus is the same in both instances. This is not the case. Execution of the following movements will readily demonstrate that whereas in abduction to the vertical the humerus rotates fully externally, *in flexion of the arm to the vertical, the bone rotates fully internally.*

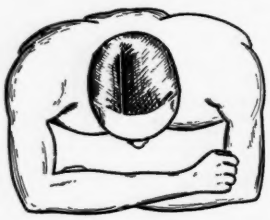

FIG. 302.—Arms and forearms horizontal, elbows flexed.

1*a*. Hold the arms vertically above the head, the elbows flexed to a right angle (*Fig.* 300, C); now bring them down to the horizontal position, i.e., right-angled abduction (*Fig.* 300, B). Note that in this position no further external rotation of the humerus can occur, indicating that at the vertical the humerus is fully rotated out.

1*b*. From the same position as in (1*a*) (vertical with elbows flexed), bring the arms down in the forward direction to the horizontal (*Fig.* 302). Note that in so doing no rotation of the humerus occurs, as indicated by the position of the lateral epicondyle being the same before and after the movement. Now rotate the forearms out (*Fig.* 303)—they readily go to the vertical—indicating that they have rotated outwards through a right angle, and that in the vertical position the humerus is fully rotated in.

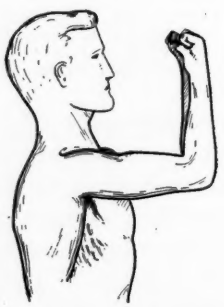

FIG. 303.—The same as *Fig.* 302 but with the forearms rotated outwards to the vertical.

Here then is an apparent *reductio ad absurdum.* When the arm is vertical no rotation is possible in the shoulder-joint, yet at the end of abduction (dependent on full external rotation) the lateral epicondyle points straight back. The explanation is simple, and is due to the fact that the one movement—i.e., flexion—is carried out in a plane a quarter of the circumference of a circle (90 degrees) in front of the plane of the other movement, abduction.

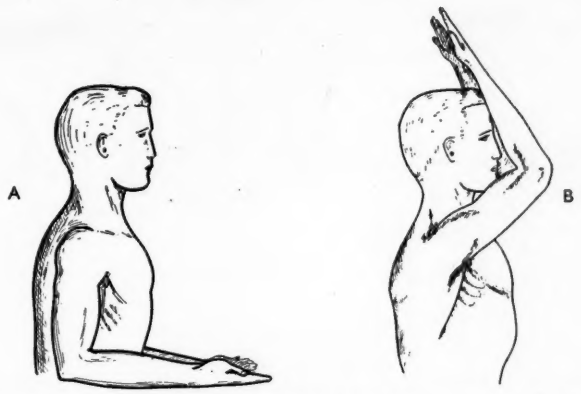

FIG. 304.—Showing that with the humerus rotated out the arms cannot be flexed to the vertical.

2*a*. Hold the elbows against the body, the forearms bent to a right angle on the arms and pointing forwards (*Fig.* 304, A). Now flex the arms by carrying them straight up in a forward direction. They can be carried up only to a right angle and a half because the humeri are rotated out (*Fig.* 304, B). The arms can be forced further up, but only by increasing the lumbar lordosis and tilting the shoulders.

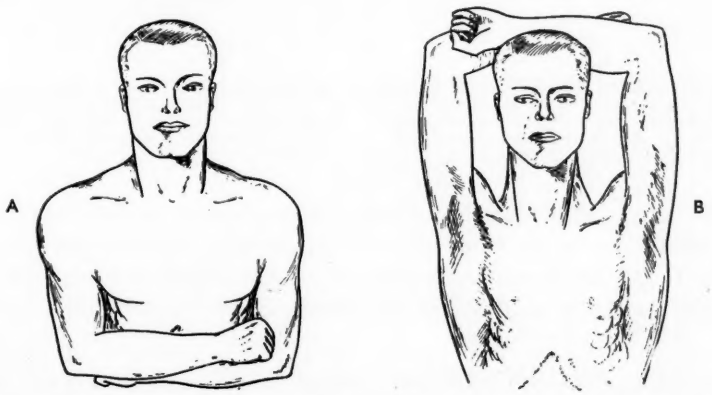

FIG. 305.—Showing that with the humerus rotated in, flexion to the vertical is possible.

2*b*. Holding the arms to the side, the elbows flexed to a right angle, and the forearms lying against the abdomen (*Fig.* 305, A), now flex the arms: they pass without effort to the vertical position (*Fig.* 305, B), because the humeri are fully rotated in.

Explanation.—The reason why internal rotation is essential to full flexion is as follows: when the arm is flexed in external rotation, the lesser tuberosity

of the humerus covered by the subscapularis impinges against the costocoracoid ligament and can only roll under this obstruction by rotating in, and then flexion can be completed (*Fig.* 306).

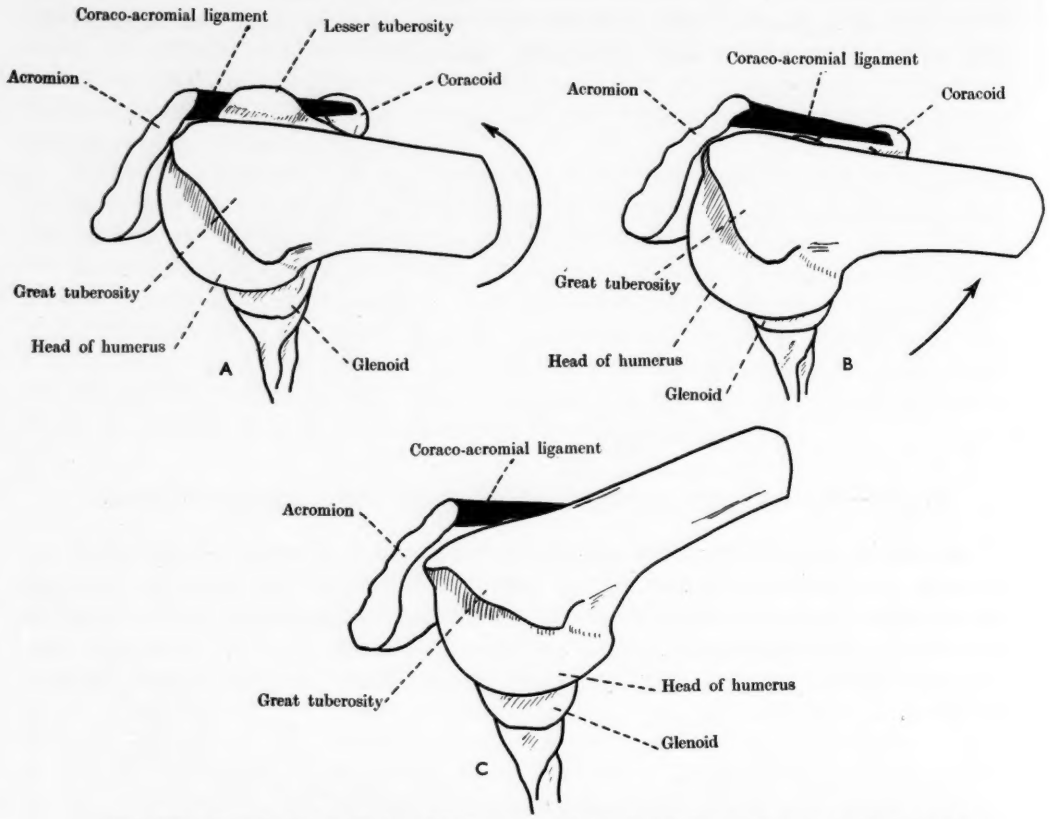

FIG. 306.—Showing that in flexion of the humerus to the vertical the lesser tuberosity impinges against the coraco-acromial arch, and the movement can only be completed when the tuberosity glides under the arch by rotating the humerus medially or inwards. A, In right-angled flexion the lesser tuberosity is obstructed by the coraco-acromial ligament when the arm is rotated out; B, With the arm rotated in, the tuberosity slips beneath the ligament; C, Further flexion is now possible.

The fact being established that flexion of the arm is as dependent on internal rotation as abduction is on external rotation, certain other important deductions can be made: (1) As to the mid-position of the humerus; and (2) As to the rotation of the humerus in different positions of the upper limb.

IV. THE MID-POSITION AND THE ROTATION OF THE HUMERUS

The Mid-position.—Holding the arm in 90 degrees abduction (horizontal in the frontal plane), the elbow bent to a right angle, the forearm directed forwards, observe that very little further abduction is possible (*Fig.* 307, A). Now bring the whole upper limb forward a few degrees—note that the limb can be elevated higher (*Fig.* 307, B). As the limb swings forward it can still be further elevated, until finally with the arm pointing straight forward and the elbow held at right

angles in front of and parallel to the transverse diameter of the thorax, the arm can be elevated to the vertical (full flexion) (*Fig.* 307, C).

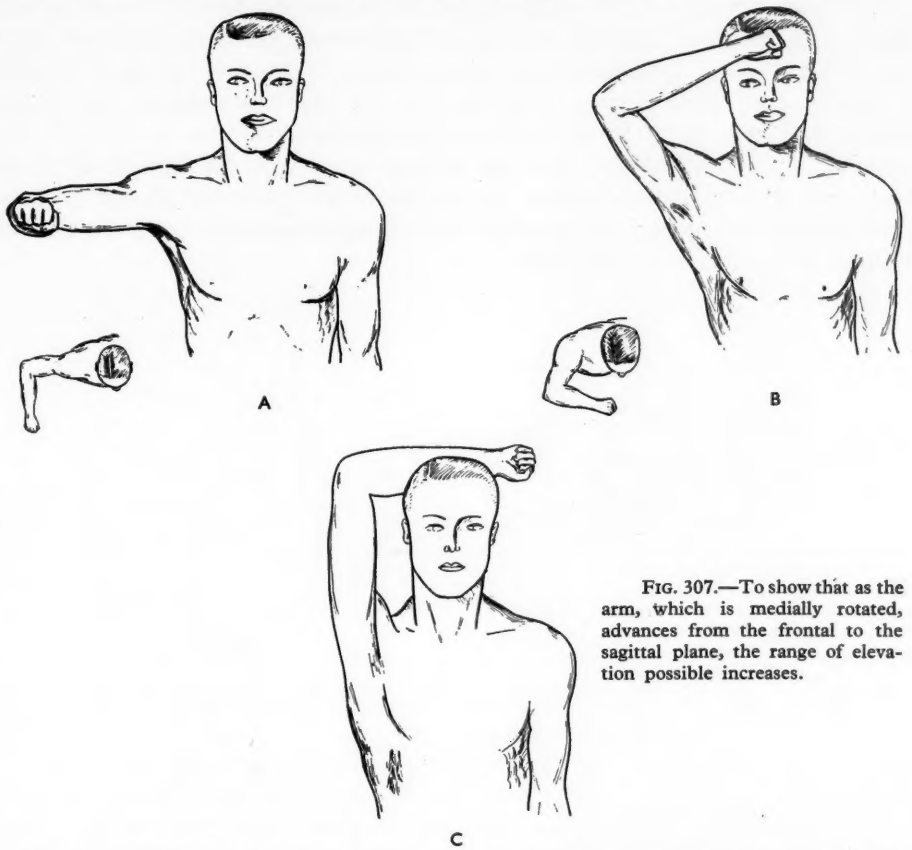

FIG. 307.—To show that as the arm, which is medially rotated, advances from the frontal to the sagittal plane, the range of elevation possible increases.

In the mid-position between right-angled abduction and right-angled flexion, the humerus can be elevated to a right angle and a half, and this range remains

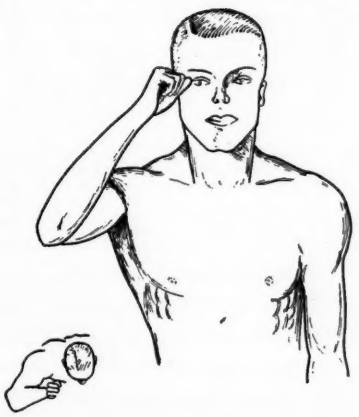

FIG. 308.—The mid-position of the humerus. This is the optimum position for the treatment of most lesions at the shoulder-joint.

the same whether the bone be rotated in or out. This is therefore the mid or neutral position, and it may be obtained with the humerus in any position of rotation (*Fig.* 308).

Rotation of the Humerus in Different Positions of the Upper Limb.—

a. When the arm is vertical no rotation of the humerus is possible (*Fig.* 309, A).

b. When the arm is at right-angled abduction or flexion, rotation of the humerus can take place through a range of just over a right angle (*Fig.* 309, B).

c. With the arm hanging at the side rotation is possible through a range of one and a half right angles (*Fig.* 309, C, D). In no position can rotation through two right angles be obtained at the shoulder.

It is then established that with the arm at the vertical no rotation is possible, but as soon as the limb begins to move downwards (whether in the frontal or sagittal plane) rotation becomes possible, and its range increases until the maximum is attained with the limb dependent.

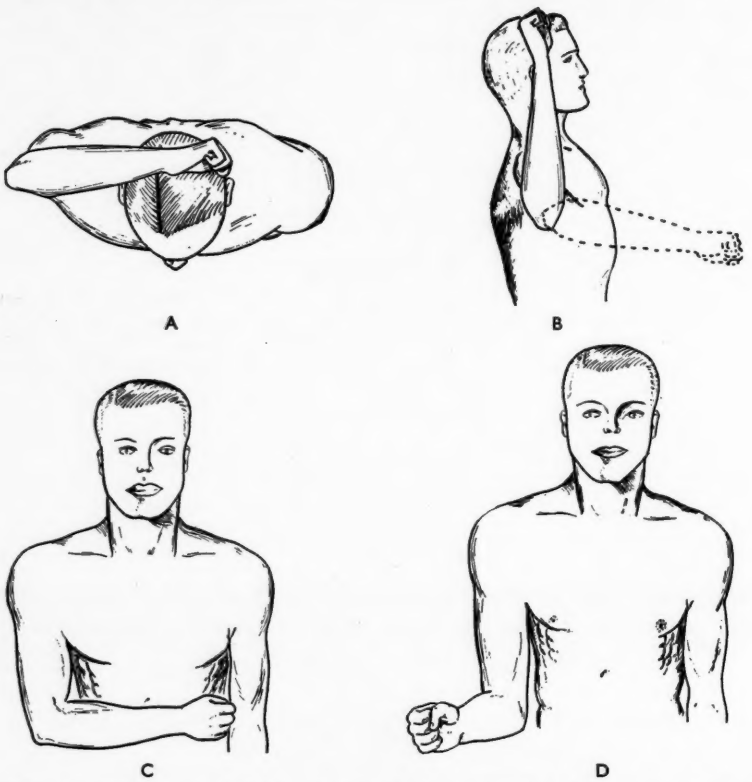

FIG. 309.—A, With the arm in full (vertical) abduction, no rotation of the humerus is possible—i.e., the joint is locked in so far as rotation is concerned; B, With the arm at a right angle, the range of rotation is shown by the interrupted line; C and D show the range of rotation with the arm at the side.

Explanation.—As pointed out above, in full abduction the humerus rotates out, the great tuberosity rotating under the acromion; so too in full flexion the small tuberosity rotates under the coracoid process. Each of these final positions is therefore a 'locking-home movement', and rotation is impossible until the joint is unlocked. Once the limb commences to move downward rotation begins at once. It would appear that this fact is an unanswerable argument *in support of the contention of Cathcart*[9] *and Lockhart*[10] *that the humerus moves on the scapula throughout the full range of abduction and flexion and not only in the first* 90 *degrees of these movements.*

In considering the movements of flexion and abduction at the shoulder in our daily activities it is very soon evident that a position which is very seldom made use of is that of right-angled abduction in the frontal plane combined with external rotation, or any degree of abduction beyond a right angle. The position may be used by some in serving at tennis or in stretching when sleepy. In doing her back hair, a woman rotates her arm out, but the position of the humerus is nearer the mid or neutral one than that of right-angled abduction (*Fig.* 310). In reaching for something on a shelf—e.g., a book—the arm goes up in the neutral position. Even in bringing the hand to the mouth the humerus is in a plane mid-way between flexion and abduction (*Fig.* 311).

FIG. 310.—To show that in doing her back hair a lady's arm is in the mid-position.

FIG. 311.—Position of the arm in carrying the hand to the mouth in eating.

A consideration of the actions performed daily will show that human beings seldom use the movements of pure flexion or abduction, but move the humerus in some plane between these two extremes, the most generally useful being a plane about mid-way between the two. This section may be concluded with the statement that the movement of abduction in the frontal plane plus full external rotation of the arm is an action which is seldom required and which could be dispensed with with little resultant loss to the efficiency of the limb, providing that elevation of the arm could be performed in a plane anterior to that of the transverse axis of the body. If the arms are held in right-angled abduction and full external rotation for a few moments it is readily appreciated that the posture is forced and uncomfortable.

V. SURGICAL APPLICATIONS

The diseased or injured shoulder if neglected takes up a position of adduction and internal rotation. Such a limb is of little use to its owner. To prevent this disability occurring it is everywhere recognized that in the treatment of such cases the arm must be abducted from the side. Some authors advise that all injuries of the joint (reduced dislocations excepted) should be treated in abduction and external rotation. In fact the position of right-angled abduction with full external rotation receives the widest acceptance as the optimum position for the treatment of lesions of the shoulder-joint which are not considered likely to end in ankylosis.

Jones and Lovett,[11] discussing the treatment of sprains of the shoulder, state: "In the case of the shoulder, the grave after-effects are due to limitation of abduction and internal or external rotation. For this reason, and because the removal of the weight of the arm expedites recovery, and because the possibility of adhesions is minimized, the arm should be treated in the abducted position, and preferably externally rotated by means of an abduction or platform splint." Quotations giving the same advice may be made from many text-books. The medical student will answer without hesitation that shoulder-joint injuries must be treated in right-angled abduction and full external rotation. A method of treatment which receives such powerful and general support as that quoted must have strong arguments in its favour. Effort will be made to show that the advice is nearly correct, but that it may be improved in some degree with vast increase in the comfort of the patient.

The Disadvantages of the Position of Right-angled Abduction with Full External Rotation.—Because of the peculiar anatomical features of this joint, particularly the osseo-fibrous arch which overhangs it, and the large important tendons and muscles which lie on or are incorporated with the joint capsule, it is readily appreciated that there is only just room normally for the execution of the complex movements of the humeral head beneath the overhanging arch. When the joint is sprained, for example, as with the tearing of muscle or tendinous fibres, there is an infiltration in and around the joint capsule, so that movement at the joint causes pressure by the over-hanging arch on exquisitely sensitive structures. This pain increases as the pressure increases. Pressure is greatest as tendons attached to the tuberosities pass under the coraco-acromial arch in the position of right-angled abduction. External rotation introduces the added factor of tension on the ligaments on the front of the joint and the medial rotators such as the subscapularis. Were a deliberate effort made to choose the position of greatest discomfort, it is probable that the position of right-angled abduction in the frontal plane with full external rotation would be the position.

Is this position more uncomfortable than may be accounted for by the combination of injury plus fixation of a joint which has hitherto been normal? In answer to this question: Two doctors practising in Johannesburg sustained injury to the shoulder and were 'put up' in right-angled abduction. Each stated that the pain was intolerable, and that not even the fear of subsequent disability could induce them to continue wearing the splints. In private and hospital practice it is similarly found that many patients will not tolerate this position after acute joint injuries. Sir Robert Jones,[12] speaking of splints to maintain abduction at the shoulder, says: "Of these none has proved satisfactory, for no matter how comfortable the splint may seem when the surgeon tries it on himself, it is a very different thing when put on a patient with a painful shoulder-joint". The fact that some, particularly hospital, patients will wear aeroplane splints for weeks is a remarkable example of human fortitude. It is probable that apart from the rigid, unalterable fixation of the joint, it is the forced unnatural position entailing pressure and tension which causes such torture to the patient.

After fractures of the surgical neck of the humerus, the upper limb is often put in extension on a Thomas arm splint in right-angled abduction and external rotation. Such a patient is nothing like so uncomfortable as the one who wears an aeroplane splint, and this for several reasons. The extension in itself probably

acts as a distraction force to those factors making for tension under the coraco-acromial arch, and, furthermore, the patient can move his body in relation to the splint, at the shoulder, and thus avoid rigid immobilization; moreover, the injury is distal to the joint capsule, and the damage done so considerable that extravasated blood may track for some distance and relieve pressure. In cases of paralysis around the shoulder an aeroplane splint is readily tolerated. Here the matter is entirely different. Owing to paralysis the head of the humerus may fall away from the coraco-acromial arch, perhaps not very much, but enough to avoid any element of tension arising.

It has been shown that right-angled abduction in the frontal plane with full external rotation is a position almost never used naturally, that it is a forced position,

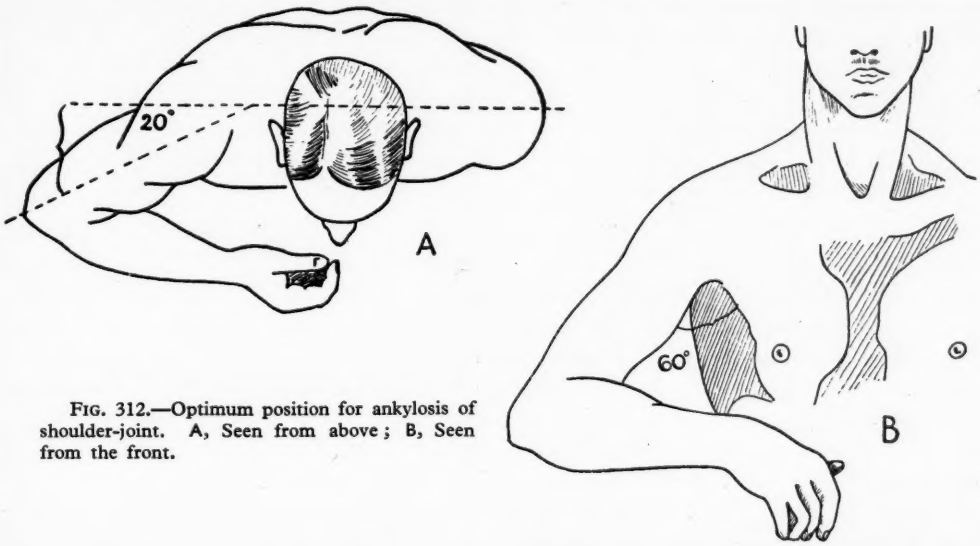

FIG. 312.—Optimum position for ankylosis of shoulder-joint. A, Seen from above; B, Seen from the front.

and that its loss would result in little if any disability. If, therefore, a position can be found which is more comfortable and which is sound from the view-point of subsequent function, that will be the optimum position for the treatment of acute shoulder injuries.

It must be made clear that this communication is not concerned with the optimum position for *ankylosis* at the shoulder, but with that position which will combine minimal discomfort with maximal subsequent function. The optimum position for ankylosis is 45 to 90 degrees abduction and 20 degrees flexion in front of the coronal plane (*Fig.* 312). The younger the patient the greater the abduction.

VI. THE OPTIMUM POSITION FOR TREATMENT OF ACUTE INJURIES OF THE SHOULDER-JOINT

The desiderata in arriving at the best position for the treatment of such injuries are:—

1. The anti-gravity muscles—i.e., abductors—must be put at rest. These are the supraspinatus and deltoid.

2. Should a muscle be torn, the position chosen must secure relaxation of the injured muscle and consequent approximation of the torn ends. The muscles

most often torn at the shoulder are the supraspinatus tendon and that of the long head of the biceps.

3. The position must avoid adhesions in the lower dependent pouch of the joint capsule.

4. The position must be such that if stiffness occurs it will be as little disabling as possible; also it must be one which will be advantageous to start from in securing a wider range of movement.

It is submitted that on anatomical, physiological, and functional grounds this optimum position is that shown in *Fig.* 308.

Advantages of the mid-position.—

1. The arm is at right angles to the body mid-way between the position of right-angled abduction and right-angled flexion.

2. The forearm is in the mid-position between full external and full internal rotation.

In this position the supraspinatus is relaxed, so also is the biceps. Neither of the tuberosities of the humerus is engaged beneath the coraco-acromial arch; *pressure* is therefore avoided. Also the capsule and its ligaments and the rotators of the joint are relaxed; *tension* is therefore avoided.

It will often be found, particularly in the condition of 'peri-arthritis' or bleeding round the joint, and sprain of the supraspinatus, that the patient has difficulty and pain in performing elevation of the arm in the frontal plane, but can readily elevate in a plane anterior to this. This is of obvious importance in treatment.

The succession of little injuries inflicted by efforts at performing abduction in the frontal plane should be avoided. The harmful effects of such repeated minor traumata have been splendidly elaborated in Mr. Watson Jones's[13] recent brilliant exposition. The mid-position can be secured by a splint fixed to the trunk, or by plaster-of-Paris, the latter being more efficient than the former.

It is interesting to see how this position, arrived at by the study of the anatomy and physiology of the joint, accords with the opinions of some observers who have not been satisfied with the routine use of abduction in the frontal plane plus full external rotation. Dickson and Crosby,[14] discussing the treatment of peri-arthritis of the shoulder-joint, say that they find the position of full abduction has no particular advantage and is likely to add to the patient's discomfort (their figure, however, shows full right-angled abduction). They advise traction in moderate abduction and external rotation. In the discussion which ensued on the presentation of this paper abduction and external rotation were generally advised in the treatment of peri-arthritis. Brockman,[15] speaking of the abduction splint, says that it is at the best an uncomfortable instrument and requires continuous adjustment. Bankart,[16] in his book on manipulative surgery, states that, after the manipulation of the shoulder-joint in which adhesions have been formed, "there is no conceivable object in making the patient 'come round' from the anæsthetic with the arm in a position of abduction and external rotation". Nowhere in his book can I find that this position is advised. Böhler[17] strongly advises that severe sprains and contusions of the shoulder be treated with the joint in the mid-position—that is, "90 degrees abduction, 90 degrees outward rotation, and 30 to 40 degrees in front of the frontal plane". In this position, he states, all the muscles of the shoulder-joint lie in mid-position, pain disappears at once, and active movements are again possible (*Figs.* 313, 314).

Speaking generally, right-angled abduction, with varying degrees of rotation, has been and is advocated for certain nerve lesions, severe sprains and contusions of the shoulder, certain tendon ruptures, and some fractures round the joint. The mid-position may with advantage replace right-angled abduction in the frontal plane in all these lesions, for the reason that it is much less irksome and that it secures better muscle relaxation. Böhler[18] strongly advises the mid-position for fractures of the surgical neck of the humerus with displacement, as in this position the pectoralis major muscle is relaxed and does not tend to pull the reduced fragments into faulty position.

FIG. 313.—Position advocated by Böhler for the treatment of severe sprains of the shoulder and of fracture of the surgical neck of the humerus. (*After Böhler.*)

FIG. 314.—Figure showing faulty position for the treatment of fracture of the surgical neck of the humerus. (*After Böhler.*)

When, however, traction is necessary in the treatment of fractures round the shoulder, it may be more convenient to apply extension on a Thomas abduction splint in the frontal plane. For reasons which have been given above this is a much less irksome form of splintage than that obtained by an aeroplane splint. It would be difficult to apply heavy traction to the humerus in the mid-position, as the extension would be awkward to secure in this plane and the counter-extension equally difficult. So, too, with some cases of fracture of the great tuberosity of the humerus with displacement, reduction might be better secured by the use of a Thomas abduction splint.

VII. CONCLUSIONS

The distinctive and unique features of the shoulder-joint are :—

1. The fact that it is overhung by a closely applied osseo-fibrous arch.

2. The architecture of the joint is such that there is only just room for joint movements beneath this arch. Any peri-articular swelling, such as follows a sprain or contusion, means that tension and pressure occur in those movements during which the humeral tuberosities slide beneath the coraco-acromial arch, the result being pain in the execution of such movements.

3. Rotation of the humerus is an essential part of the movements of abduction and flexion above the horizontal plane ; so much so that abduction above 90 degrees cannot be executed unless the humerus is fully rotated out, and the last 50 degrees of flexion are equally dependent on internal rotation.

These features distinguish the mechanics of the shoulder-joint from all other joints ; true, lateral flexion of the spine cannot occur without rotation : however,

not one, but many joints are concerned in this action; rotation of the femur when the psoas flexes the hip-joint is the nearest analogy in the body, but here the joint architecture is of a much simpler type than at the shoulder. Rotation being essential to the free movements at the joint, the correct appreciation of this phase of joint activity becomes of paramount importance in the superintendence of cases of disease or injury at the shoulder. As the range of rotation of the humerus is at its maximum with the arm at the side of the body, it is reasonable to instruct the physio-therapeutist to exercise the rotary function of the joint with the limb in this position, more especially as the sufferer will tolerate this movement better here than with the limb elevated. Abduction in the frontal plane, particularly if combined with full outward rotation of the arm, is an extremely uncomfortable position for a recently injured shoulder; moreover, it is a position which is of hardly any economic value, and should therefore be discontinued as a method of rigid fixation. In cases of peri-arthritis, the mid-position is that which is indicated on anatomical and physiological grounds, and is, furthermore, a position which can be assumed or maintained with far less discomfort.

VIII. SUMMARY

1. Full outward rotation of the humerus is essential to the performance of full abduction (Martin).

2. Full inward rotation of the humerus is essential to the performance of full flexion.

3. With the arm vertical no rotation of the humerus is possible. The degree of rotation increases from above down and is maximal with the arm at the side.

4. Horizontal abduction in the frontal plane combined with full external rotation of the arm is a movement seldom if ever used.

5. The position defined in (4) is one which causes extreme discomfort.

6. Fixing the humerus in this attitude in injuries at the shoulder should therefore be discontinued.

7. The mid-position is that in which the joint should be splinted. It is anatomically and physiologically sound and much less irksome.

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MALLET FINGER

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It is the purpose of this paper to describe a rational method of treatment of the mallet or drop finger, and, at the same time, record the conclusions which have resulted from the experience of treatment in a series of 35 consecutive cases.

ETIOLOGY

Rupture of the extensor tendon at its insertion into the base of the terminal phalanx is the commonest of the subcutaneous tendon ruptures, and it was noted in the series under review that the injury was encountered once in 900 cases at a large Surgical Out-patient Department. The rupture is produced by indirect injury, the actively contracting tendon being subjected to a sudden passive flexing force, or less commonly to direct violence, the tendon being nipped against the dorsal aspect of the base of the phalanx. It is thus common in cricket, football, and baseball players ; but such an apparently trivial accident as might occur during the making of a bed is frequently recorded as the cause of the injury. The history of having heard or felt the tendon snap and having experienced a sharp pain in the region of the distal interphalangeal joint is frequently given, but if the hand has been cold, the condition of the finger may not be noticed until some time afterwards.

SYMPTOMS

The injury is often diagnosed as a stave, occasionally as a dislocation, and strapped or splinted accordingly, and it is only on the removal of the dressing that it is realized that dorsiflexion is no longer possible. As seen shortly after the accident, there is swelling and tenderness especially on the dorsal aspect of the interphalangeal joint, the swelling tending to mask the flexion of the terminal phalanx which is present. Complaint is made of a dull, aching pain which is transformed into a sharp stab when the injured tendon is inadvertently contracted along with the uninjured extensors. Active extension in the complete lesion is impossible.

CLINICAL TYPES

Four separate clinical types can be recognized :—

Type 1.—Incomplete loss of extension due to a partial tear or stretching of the expansion. There is no demonstrable radiological lesion.

Type 2.—Absolute loss of extension, due to a complete tear in the expansion, but with no demonstrable radiological lesion (*Fig.* 315).

Type 3.—Absolute loss of extension with a demonstrable radiological lesion consisting of a chip of bone, often triangular in shape, separated from the base of the phalanx (*Fig.* 316).

Type 4.—A type seen in children—often the result of extreme violence—consisting of separation of the epiphysis at the base of the phalanx (*Fig.* 317). This type may be compound.

TREATMENT

A vast number of methods of treatment are in existence, most of which attempt by means of metal, wood, or celluloid splints to keep the terminal phalanx in extension. They have as obvious disadvantages imperfect immobility, clumsiness, tendency to pain from pressure, ease of removal by the patient on the slightest

FIG. 315.—*Type* 2. Showing flexion of the terminal phalanx but with no demonstrable radiological lesion.

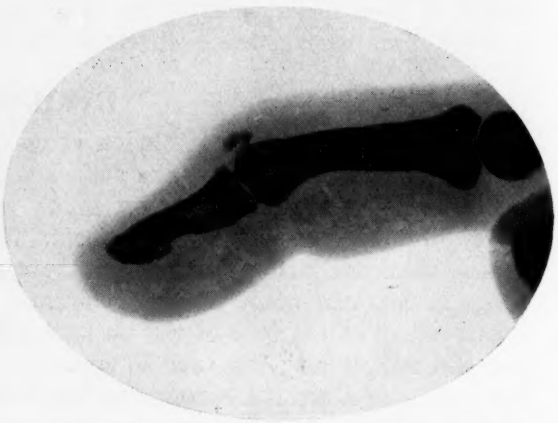

FIG. 316.—*Type* 3. Showing the chip of bone separated from the base of the terminal phalanx.

discomfort, complete disability of the hand during the whole period of treatment, and the fact that the position is anatomically unsound from the point of view of healing, the central slip of the extensor being liable to tension when the sound fingers extend.

FIG. 317.—*Type* 4. Showing separation of the epiphysis.

FIG. 318.—*Type* 4. In plaster after reduction.

The most reasonable position for healing must be right-angled flexion at the proximal interphalangeal joint with hyperextension at the terminal interphalangeal joint, for in this position active extension is impossible, the central slip of the tendon being relaxed.

To obtain this position, securing at the same time absolute immobility with the minimum loss of function in the hand during treatment, a plaster case is ideal. The method eventually evolved for the application of the plaster case is as follows :—

A piece of Cellona plaster bandage about two feet long is rolled into a tube of such inside diameter as to roughly fit the finger. The tube should be rather longer than the finger to be treated, and should be cut on the slant at one end, so that it may fit closely at the web (*Fig.* 319).

The patient is shown how to press the thumb against the palmar surface of the terminal phalanx so that the proximal interphalangeal joint is a right angle and

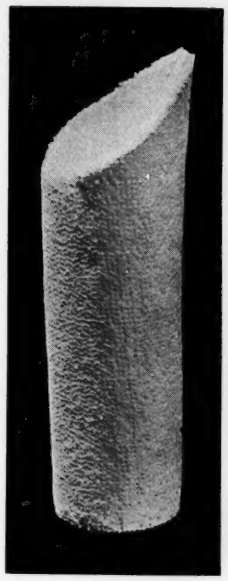

FIG. 319.—The plaster tube.

FIG. 320.—The position in which the patient is instructed to hold the finger.

the terminal phalanx hyperextended. A few moments spent on instruction are not wasted (*Fig.* 320).

The finger is now inserted into the dry tube of plaster (*Fig.* 321) and the hand dipped momentarily in warm water. The finger with its wet plaster covering is then squeezed tightly in the hand of the surgeon, and the patient is instructed to take up the required position until the plaster dries (*Fig.* 322).

If this technique is followed accurately, the operator especially refraining from holding the finger in position himself, no trouble will result from pressure, and in this series no complications have arisen other than the breaking of a plaster from a fall on ice and the return of four patients to have the splint replaced, being under the erroneous impression that plaster-of-Paris is waterproof. Many report their ability to carry on their normal occupations, even to the driving of a heavy motor lorry.

At the end of the period of immobility, which should be not less than five weeks, the case is removed by soaking in water to facilitate the straightening out of the right-angled bend, after which withdrawal of the finger is easy. Some cases show immediate full dorsiflexion, while others, although showing active extension against resistance, are still some degrees short of the normal. No estimate of the end-result should be made until about three weeks have elapsed, when with use many cases will be found to have regained the normal range of movement. No trouble has been experienced with persistent stiffness at the proximal interphalangeal joint, and it is probable that the rapid mobilization of the joints in the hands of

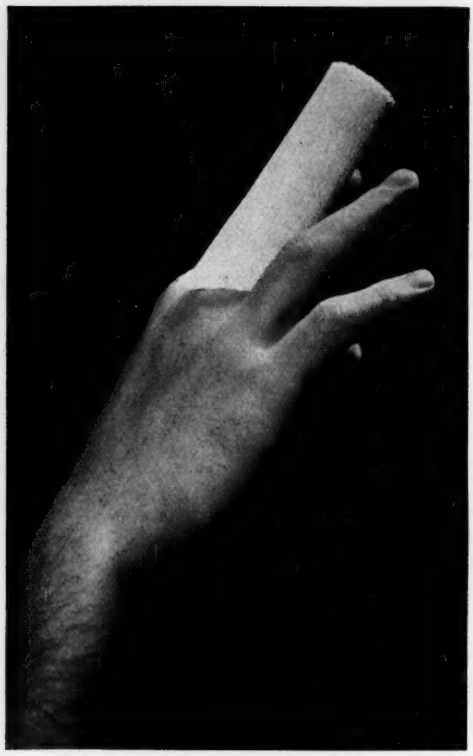

FIG. 321.—The dry plaster tube applied to the finger.

FIG. 322.—The finger held in the required position until the plaster dries.

a masseuse is inadvisable, as the scar tissue is still stretchable, and full passive flexion of the terminal phalanx predisposes to a poor result.

PROGNOSIS

Prognosis depends on: (1) The time which has elapsed since the injury; (2) The age of the patient; (3) The type of injury.

With regard to the type of injury, *Type* 1 gives excellent results without deformity. *Types* 3 and 4 can be relied upon to give good results, but with some residual thickening at the terminal interphalangeal joint. The results in *Type* 2 vary, so that the prognosis must be more guarded, for although the joint is of necessity opened into in *Types* 2 and 3, in *Type* 2 there is always the possibility

DETAILS OF 35 CASES OF MALLET FINGER

INITIALS	AGE	SEX	FINGER	DAYS SINCE INJURY	X RAYS	TYPE	RESULT OF TREATMENT
I. S.	26	M	R 3	5	Pos.	3	Full extension
J. McL.	13	F	R 3	11	None	2 or 3	Extension slightly less than other side
Mrs. L.	49	F	R 3	1	None	2 or 3	Very few degrees of full extension
I. S.	22	F	L 4	5	Pos.	3	Full extension
J. B.	13	M	L 4	4	Neg.	2	Few degrees short of full extension
Mrs. M.	40	F	R 3	35	Pos.	3	Active extension but not full
P. S.	24	M	R 3	150	Neg.	2	Put up in plaster as experiment; no indication for operation; definite active extension obtained
Mrs. P.	33	F	L 4	7	None	2 or 3	Extension almost full
S. P.	21	M	L 2	2	None	2 or 3	Full extension
L. A.	29	M	L 3	14	Neg.	2	Strong active, but incomplete, extension
J. G.	50	M	R 4	9	Pos.	3	End-result unknown
Mrs. B.	34	F	R 3	28	Neg.	2	*Operation*: Good result, but extension a little short of full
Dr. B.	36	M	R 4	5	Neg.	2	Complete extension
J. A.	12	F	L 4	2	Neg.	2	Complete extension
Mrs. S.	65	F	R 3	?	Neg.	3	Extension almost full
B. O.	14	F	R 3	24	Neg.	2	A few degrees short of full extension
Mrs. M.	35	F	R 4	21	Neg.	2	Full extension
J. F.	24	M	R 3	21	None	2 or 3	End-result unknown
J. B.	31	M	L 2	7	Neg.	1	End-result unknown
A. M.	20	M	L 5	1	Pos.	3	Full extension
R. A.	42	M	R 5	7	Neg.	2	End-result unknown
W. McL.	21	M	L 4	28	Pos.	3	Full extension
C. B.	?	F	R 2	28	Neg.	2	Some improvement in function
R. B.	36	M	R 5	36	Neg.	2	Strong active extension, but not quite full
F. T.	41	M	R 5	28	Neg.	2	Patient did not wish treatment
H. T.	23	M	L 5	28	Neg.	1	Full extension
N. L.	35	M	L 5	12	Neg.	2	*Operation*: Following this, patient removed plaster and dressing; joint became infected; end-result—ankylosis in semiflexed position
T. H.	12	M	R 2	3	Pos.	4	Compound injury—operation—perfect result
D. L.	12	M	R 4	1	Pos.	4	Perfect result
Miss T.	47	F	R 5	21	Neg.	2	Strong active extension, but not quite full
M. L.	13	M	R 5	28	None	2	*Operation*: Full extension
R. G.	38	M	R 2	2	Neg.	2	Strong active extension, but not full
M. C.	61	F	R 2	2	Pos.	3	Not quite full extension
J. S.	15	M	R 4	12	Pos.	4	Perfect result
J. D.	18	M	R 2	35	Pos.	3	Conservative treatment; some active extension obtained
J. D.	18	M	R 3	35	Pos.	3	*Operation*: Almost but not quite full extension—very much better than finger treated conservatively

that the torn end of the proximal portion of the tendon has turned into the joint through the rent in the capsule, predisposing to disappointing results.

OPERATIVE TREATMENT

Lack of uniformity in the results obtained with conservative measures in the complete lesion without fracture makes operative measures worthy of consideration; but as healing with some degree of dorsiflexion can always be obtained by conservative treatment, it is evident that ample justification for operation is only present where occupation or æsthetic considerations demand that a chance of a perfect result be offered.

Old Cases.—For the reasons outlined above, many patients do not report till some weeks after the injury, and it will be seen from the Table that a considerable proportion of cases fall into this category.

During the five weeks following the injury, some improvement in function and deformity may be expected by immobilization in plaster as in the fresh case, so that indications for operation are essentially as for the fresh case falling into *Type* 2.

Indications for Operative Treatment.—It follows from the above that operation should be reserved for :—

1. Compound injuries—often epiphysial separations, and referred to here as *Type* 4.
2. Certain fresh cases belonging to *Type* 2 where at least a chance of full dorsiflexion is essential.
3. Certain old cases which demand a chance of increased dorsifiexion.

The Operation.—The success of the operation depends on the use of a plaster case applied in exactly the same manner as in the fresh injury; hence the necessity for instructing the patient in the method of application and retention and the use of local infiltration anæsthesia at the base of the finger.

The compound injury is treated by the usual methods. Where the diaphysis has been torn away from the epiphysis the base of the nail has probably been evulsed and will require removal. No sutures are inserted in the capsule or extensor expansion, apposition of the torn edges being maintained by hyperextension alone.

The best incision for the deliberate operation has been found to be L-shaped, the short limb crossing the dorsum of the finger transversely, just proximal to the base of the nail, the long limb being dorsi-lateral, extending in a proximal direction (*Fig.* 323). This incision gives good exposure, while the sloughing which often follows the use of the U-shaped incision, and the disfiguring and sensitive scar which follows the midline dorsal incision, are avoided.

While the skin-flap is being dissected up, care must be taken not to damage the capsule or the remanants of the torn expansion. Procedure now will depend on the type of case. In the fresh case where the proximal end of the torn expansion has turned into the joint, the edges should be approximated, and two sutures of 0000 catgut mounted on the smallest usable curved needle inserted. If suture is impossible, as it often is, the edges should merely be approximated and the terminal phalanx maintained in hyperextension.

In the old case a previous chip fracture or tear may be reproduced and the edges freshened, suturing where possible, otherwise maintaining the edges in apposition

by continuous hyperextension. When a few sutures of fine catgut or waxed silk have been inserted into the skin edges, the wound is sealed with a single layer of gauze soaked in collodion.

A sterilized plaster tube is now slipped over the finger with care to maintain the hyperextension, and the hand is dipped in sterile water. When the excess water has been squeezed from the plaster in the surgeon's hand, the patient is given control

FIG 323.—The incision.

and holds the finger in the desired position till the plaster is set. The sutures need not be removed until the plaster case is discarded at the end of five weeks.

I am deeply indebted to Mr. T. McW. Millar and Mr. D. Stewart Middleton, of the Surgical Out-patient Department of the Royal Infirmary, Edinburgh, both for the kindness they have shown in permitting me to treat their cases and for their sustained interest and advice.

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BLOOD-PRESSURE DURING SPINAL ANÆSTHESIA

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THE article by Miss Joan Walker in the July issue of the BRITISH JOURNAL OF SURGERY on the use of hypertonic saline for the purpose of raising the blood-pressure, tempts me to record many facts and experiences similar to those she has noted, but to offer a different interpretation of those facts, and a different treatment for the conditions met with—a treatment, moreover, which has had a considerable measure of success. This is not in any way to decry the treatment of low blood-pressure by hypertonic saline, of which I have had no experience, but merely to show that many cases of low blood-pressure need no special treatment, that low blood-pressure and 'shock' are by no means synonymous terms, and that low blood-pressure when it requires treatment can frequently be remedied by other and somewhat less heroic measures.

Confining our attention for the moment to abdominal sections performed under spinal anæsthesia, one is struck by the fact that they divide themselves into two very definite and distinct groups: (1) Cases in which the blood-pressure falls at once and steeply before the peritoneum is opened; and (2) Cases in which the blood-pressure either rises or remains steady after the preliminary ephedrine and spinal anæsthetic have been given, and usually but not always falls when intra-abdominal manipulation such as exploration or the packing of gauze is undertaken.

The next point to notice is that the cases that show a steep and immediate fall of blood-pressure when the spinal anæsthetic is given are the acute abdominal conditions with great pain. So typical is this movement of the blood-pressure in these acute cases that if it does not occur it is safe to say that the diagnosis is mistaken and that the abdominal condition is not very acute. A few days ago I was asked to give a spinal anæsthetic to a man of 52 diagnosed as a perforated duodenum. On giving the spinal anæsthetic (anæsthesia to the nipple line) the blood-pressure fell, but only 10 mm. Hg—from 120 to 110. Very thorough and persevering efforts were made to find the perforation, during which the blood-pressure fell to 75 mm. Hg, rising to 100 within five minutes of their cessation. No perforation was found, but a partial obstruction by a band of the terminal ileum. During these proceedings I ventured to suggest to the surgeon that no perforation would be found, and, further, that the abdominal condition, whatever it was, was not very acute. The actual findings were entirely in accordance with the movements of the blood-pressure. *Table I* gives the records of 20 acute abdomens opened under spinal anæsthesia.

The 20 cases have been selected from my records; they are by no means consecutive. It is pertinent to ask why select the cases if the fall of blood-pressure is so characteristic of acute abdominal conditions. The reasons are two in number. In the first place I have tried to give as great a variety of acute cases as possible, and in the second place I have tried to choose cases uncomplicated as far as possible by other conditions likely to cause a sharp fall of blood-pressure.

Table I.—Blood-pressure Records in 20 Operations Performed under Spinal Anæsthesia for Acute Abdominal Conditions

No.	Initials	Sex	Age	Disease	B.P. 1	B.P. 2	B.P. 3	Condition Found and Remarks	Result
1	A. R.	F.	50	I.O.	110	70	45–50	Two feet gangrenous gut removed ..	Recovery
2	W.	F.	40	T.O.	100	?	90–100		Recovery
3	W.	M.	54	I.O.	160	85	85–140–115	Prolapse of gut (3 ft.), foramen of Winslow	Recovery
4	D.	M.	70	I.O.	140	60	60–90	Mesenteric thrombosis (2½ ft. gut) ..	Died (10 days)
5	E. H.	F.	—	I.O.	170	100	90–140		Died (3 weeks)
6	H. D.	M.	—	P.D.	120	63	?–50	Perforation 12 hours old	Recovery
7	A. B.	M.	53	P.D.	140	75	80–65		Recovery
8	N.	F.	71	I.O.	145	90	90–112–100	71 years old. Very fat	Died (17 days)
9	A.	M.	—	A.A.	120	70	64–90	Very extensive peritonitis	Recovery
10	A. L.	M.	56	A.A.	120	?	60	Very extensive peritonitis	Recovery
11	E.	F.	44	I.O.	110	60	60–80		Died (16 days)
12	A. M.	M.	63	S.I.H.	150	80	70–92–85	Very ill. Bowel of doubtful vitality. Chronic bronchitis and emphysema ..	Died (4 days)
13	K. O.	F.	—	P.G.	120	?	?–105	2½ pints saline intravenously	Died next day
14	W.	F.	46	P.D.	120	60	60–60		Recovery
15	B.	F.	46	I.O.	110	?	?–70	Richter's hernia	Recovery
16	C.	F.	66	I.O.	140	?	?–110	66 years old	Recovery
17	B.	—	62	S.U.H.	180	?	?–80	Very great pain	Recovery
18	T. H.	M.	40	P.D.	130	75	60–120		Recovery
19	H.	F.	66	S.F.H.	120	75	64–78		Recovery
20	H. K.	F.	50	A.P.	180	70	70–60	Very ill	Died (4 days)

I.O. = Intestinal obstruction
P.D. = Perforated duodenum
T.O. = Twisted ovarian pedicle
A.A. = Acute appendicitis
S.I.H. = Strangulated inguinal hernia
P.G. = Perforated gastric ulcer
S.U.H. = Strangulated umbilical hernia
S.F.H. = Strangulated femoral hernia
A.P. = Acute pancreatitis
B.P. 1 = Blood-pressure before spinal anæsthetic
B.P. 2 = Blood-pressure after spinal anæsthetic
B.P. 3 = Blood-pressure during operation

Amongst these other conditions that are frequently met with in the course of one's usual routine are the following: (1) Old age; (2) Anxiety or extreme nervousness; (3) Want of tone, as in the fat and flabby; (4) Overdosage with sedative drugs—paraldehyde, omnopon, and scopolamine; (5) A high blood-urea indicative of a toxic state; (6) The severely dehydrated. Any of these conditions, singly or in combination with each other or with an acute abdominal lesion, will cause a steep fall of blood-pressure within a few minutes of the exhibition of a spinal anæsthetic before the peritoneal cavity is opened. With the exception of (5) they are all fairly obvious and should not cause confusion. Still there are many patients who come with acute abdominal lesions who are neither fat nor flabby, nor severely dehydrated, nor overdosed with sedatives, who are still young and otherwise healthy, and not extremely nervous. It is in such cases that the steep fall of blood-pressure as the immediate result of a spinal anæsthetic is characteristic of an acute abdominal lesion.

The essential feature about all these cases is pain. Some were suffering from shock. Perhaps one ought to say that all were suffering from shock, but in such varying degrees that clinically there was the most marked difference between *Case* 4, an old man with mesenteric thrombosis, and *Case* 9, a man of 45 in comparatively good condition.

The next point to note is that the relief of pain and the fall of blood-pressure occurred simultaneously, both dramatic events. The conclusion is almost irresistible that the spinal anæsthetic caused both, and by the same action. There is no doubt that the pain is relieved by the action of the spinal anæsthetic on the posterior roots, and it seems almost certain that whether pain and pressor impulses are the same and carried by the same fibres or not, both are carried in the posterior roots and are paralysed by the spinal anæsthetic. The vasomotor centre which has been bombarded by these pressor impulses suddenly ceases to receive them and so ceases to send out vasoconstrictor impulses, hence the fall of blood-pressure. Parsons and Gray, in 1912, pointed out that pressor impulses leading to a rise of blood-pressure were productive of the first stage of shock, and it is now well known that the viscera of animals dying from shock are pale. The intense vasoconstriction is obviously a protective mechanism for the supply of vital organs with whatever blood the organism still possesses. It is important when the spinal anæsthetic has abolished this protective mechanism that the patient should be put in a slight Trendelenburg position so that the heart shall receive an adequate supply of blood. The action of the spinal anæsthetic in causing a fall of blood-pressure is too quick to be due to paralysis of the anterior roots (vasoconstrictors) and the exudation of fluid from the vessels into the tissue spaces. One must suppose that this latter process takes some time. Holt and Macdonald found that in order to reduce their animals to a state of shock they had to lose on an average no less than 48 per cent of their calculated circulating blood-volume. It is impossible to suppose that a spinal anæsthetic can in five or ten minutes cause any comparable loss; yet if spinal anæsthesia be the cause of shock, since shock is proportional to fluid loss, this is what one must suppose.

Not only does the rapidity with which the blood-pressure falls and its coincidence with the disappearance of the pain point to the action of the spinal anæsthesia on the posterior roots as being the cause of these phenomena, but experimental evidence supports this view. If a cat be given a spinal anæsthesia

nearly but not quite up to the origin of the phrenic nerves, the blood-pressure will fall to about 60 mm. Hg. If now the abdomen be opened and traction made on the stomach, the blood-pressure will fall still further, but resumes its former level on releasing the stomach. If this traction be repeated at intervals of a few minutes it will be found that each succeeding fall is followed by a rise of blood-pressure, and that this rise becomes more marked on each occasion. It is obvious that pressor impulses which had been abolished by the spinal anæsthesia are gradually coming into play again as the anæsthesia disappears. That the anæsthesia is disappearing is proved by the fact that the base line of 60 mm. Hg to which the spinal anæsthesia had originally reduced the blood-pressure, has been gradually rising throughout the experiment.

If then we take this view of the fall of blood-pressure—namely, that it is due to the abolition of pressor impulses by the spinal anæsthesia—we shall not call it 'shock', we shall not suppose it to be due to the leakage of plasma from the circulation, and there will be no indication that this fall of blood-pressure needs any treatment at all. Whether it be desirable to institute any special treatment at the moment or not will depend entirely on the condition of the patient before the spinal anæsthesia was given. If, for example, the patient has a recent (2–3 hours) duodenal perforation, when he comes on to the operating table he will be suffering intense pain in spite of the morphia which he has in all probability received. In such a case the spinal anæsthesia will give him such tremendous and almost instantaneous relief that his condition is much improved in spite of having lost half his blood-pressure. The patient at least thinks so. No treatment is necessary.

If on the contrary the patient be severely dehydrated from any cause, then either prior to or immediately after the spinal anæsthesia he will require treatment to restore his diminished blood-volume. One such case came on to the table with a blood-pressure of only 70 mm. Hg. The spinal anæsthesia caused the radial pulse to disappear. Intravenous saline (3 pints) given slowly throughout the operation, together with the intramuscular injection of pituitrin, raised the blood-pressure to 130 mm. Hg by the time the operation was finished, and the patient made an excellent recovery. The essential state to treat here was *lack of blood*, not lack of blood-pressure, which is primarily significant as an indication of the lack of circulating fluid. It is difficult to suppose that hypertonic saline would have been a more efficient remedy in this case, and yet it is just the kind of case in which one would imagine hypertonic saline would show to great advantage.

A third point to note is that in none of these cases is the blood-pressure low before the spinal anæsthetic is given. We so often hear that the blood-pressure is low in shock that we overlook the fact that in the early stages of shock the blood-pressure is high, and that it fails in spite of a high degree of vasoconstriction only when the patient or animal has lost between 40 and 50 per cent of his blood-volume. It is obvious that in the above list no such loss had taken place. The case just referred to might possibly have done so, as his blood-pressure was only 70 mm. Hg.

For the sake of comparison, *Table II* shows a series of 20 cases of non-acute abdominal sections done under spinal anæsthesia. All these cases, like the preceding group, had 100 mg. ephedrine injected with the local anæsthetic before the spinal anæsthetic. The first ten were all done by the same surgeon, and the second ten by ten different surgeons. It will be noticed that the blood-pressure rose in 18 out of the 20, and in the other 2 remained the same, *after the injection of the spinal*

anæsthetic, before the peritoneum was opened. Subsequently, during the operation it fell in every case. Here then is a totally different picture of the course of the blood-pressure which calls for a different explanation of the fall. If spinal anæsthesia causes first a stagnation of blood so complete as to set aside a considerable fraction from the circulation, and secondly an exudation of plasma from the capillaries comparable to that seen in traumatic shock, why does it not occur in the chronic case as in the acute? I can see no answer to that question. It is certainly not due to the condition of the acute case being more serious; it may or may not be. No one would suggest that a perforated duodenum in an otherwise healthy man, for instance, was as serious as a malignant growth of the colon or the head of the pancreas, or even a condition requiring gastrectomy. The reason why we get a fall in the acute case we have already shown to be the obliteration of pressor impulses by the spinal anæsthetic. In the chronic case, on the other hand, we do not get a fall of blood-pressure because the vasoconstrictors of the upper dorsal anterior roots are not touched by the anæsthetic, but are, on the contrary, powerfully stimulated both by the ephedrine and the condition of the patient. This overaction of the upper dorsal vasoconstrictors is simply part of the means that every organism uses to adapt itself to new conditions. It does not occur in the very old, the very feeble, or the very ill, and in these cases the blood-pressure falls.

Table II.—Blood-pressure Records in 20 Operations Performed under Spinal Anæsthesia for Non-acute Abdominal Conditions

No.	Initials	Sex	Age	Operation	B.P. 1	B.P. 2	B.P. 3
1	A. F.	M.	—	Colostomy and excision ..	130	150	100
2	A. K.	M.	56	Gastro-cholecystostomy ..	134	138	60
3	D. D.	M.	65	Resection of growth of colon ..	150	150	65
4	B. O.	F.	45	Double lateral anastomosis ..	100	140	90
5	C. G.	M.	60	Gastro-enterostomy	130	130	60
6	C. B.	F.	—	Resection of growth	110	150	80
7	D. B.	F.	—	Appendix (interval)	118	123	85
8	E. R.	F.	68	Resection of growth	145	160	80
9	G. S.	M.	49	Gall-bladder..	120	160	125
10	H. M.	F.	—	Gall-bladder..	130	135	60
11	J. H.	M.	37	Gastric ligation	120	160	120
12	K. W.	M.	48	Resection of growth	120	170	80
13	M. L.	M.	35	Gastrectomy..	110	150	90
14	M. R.	M.	37	Nephrectomy	145	190	102
15	N. S.	M.	56	Gastro-enterostomy	115	130	75
16	O. A.	F.	43	Double pyosalpinx	120	140	110
17	P. L.	F.	72	Exploration for malignancy ..	140	150	60
18	R. N.	F.	40	Transverse colostomy	130	140	110
19	J. A.	M.	48	Appendix (interval)	140	170	125
20	–. S.	F.	48	Cholecystectomy	125	145	80

B.P. 1 = Blood-pressure before spinal anæsthetic
B.P. 2 = Blood-pressure after spinal anæsthetic
B.P. 3 = Blood-pressure after opening of peritoneal cavity

Why does the fall occur later on? The time at which it occurs—namely, when the exploration is made, or severe traction is made, or large swabs are being packed into the abdominal cavity—suggests very strongly that it is due to stimulation of the vagus, a widely distributed nerve untouched by the anæsthetic. The fact

that a fall of blood-pressure is more likely if the operation be on the upper abdomen than on the lower points in the same direction, as does also the animal experiment already referred to in this paper.

Miss Walker refers to the idea that the fall of blood-pressure is due to absorption of the anæsthetic into the blood-stream, but rejects it as unlikely. I agree with her, but should put it more strongly. Ten milligrams of novocain injected into the cisterna magna of a cat will kill it in one and a half minutes by paralysis of the respiratory centre. If artificial respiration be applied in time the cat will be rescued, and in a few minutes will be able to breathe by itself. If now we allow it an hour in which to destroy any novocain left in its system and then inject 20 mg. of novocain into the jugular vein, it affects neither the blood-pressure nor the respiration. Moreover in one experiment which I did with Macdonald we injected $7\frac{1}{2}$ mg. of novocain every minute for twenty-four minutes before the respiration was affected —that is, eighteen times the dose that was lethal if put into the cisterna magna. Again, I injected 300 mg. into the median cephalic vein of a patient in the course of ten minutes; the blood-pressure remained steady between 140 and 160 mm. Hg, and the respiration was unaffected. These facts have been published before; but one constantly sees this bogy of blood absorption of a spinal anæsthetic treated as a serious danger.

One other point calls for notice, this time a difference in facts observed. Miss Walker records a fall of blood-pressure in 157 cases out of 300 (52·3 per cent), and 42 failures to obtain anæsthesia (14 per cent). Excluding acute cases, and patients in whom one would be unlikely to find vigorous adaptation to new conditions—that is, the fat and flabby, the very old, or very ill—I find that in the last 100 cases of which I have records, in only 5 (5 per cent) has there been a fall of blood-pressure before the operation began, and there have been no failures to obtain the required anæsthesia and muscular relaxation. It is perhaps impossible to explain such different results, but one factor may account for part of the discrepancy. I have used the following solutions—heavy duracain (a 10 per cent novocain solution), heavy percain (a 0·5 per cent solution), and heavy decicain (a 1 per cent solution), each with a specific gravity of 1·025. These solutions are injected with the patient lying on his side, and he is turned on to his back immediately after the injection is finished. In this way more posterior roots are affected than anterior, and as it is the paralysis of the anterior roots that causes a fall of blood-pressure, I regard this technique as both more reliable and safer than the use of light solutions.

A fall of blood-pressure during spinal anæsthesia may be due to one or more of many causes. Treatment, if any, will depend on the diagnosis of the cause. Whether any of these causes is more efficiently remedied by the use of hypertonic saline, which has certain admitted disadvantages, than by more ordinary methods, seems doubtful.

PATHOLOGICAL FRACTURE OF THE HUMERUS COMPLICATING LATE SECONDARY SYPHILIS

By H. JACKSON BURROWS, London

CASE REPORT

The events in the following case are described in the order in which they presented themselves to the surgeon, as they seem more instructive in this sequence than in strictly chronological order.

Fig. 324. — The right humerus (posterior oblique projection), showing the pathological fracture.

Fig. 325.—Posterior projection of the fractured right humerus.

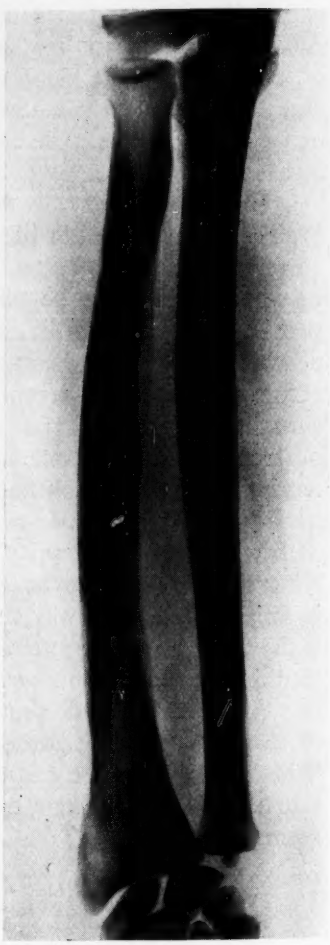

Fig. 326.—The left radius and ulna.

History given by the Patient and Clinical Examination.—The patient was a maintenance mechanic, aged 28 years. Ten weeks previously, when throwing a skittle ball with

his right arm, he heard something snap, and the arm dropped and became useless. He was treated in a hospital with a Thomas's arm bed-splint. After seven weeks he returned to work.

The day before he came under observation, he was boarding a tramcar, which started suddenly as he was hanging on with his right hand, when he experienced sudden pain in the right arm and loss of use of the limb.

On examination, any movement of the right arm resulted in pain.

X-RAY EXAMINATION.—The humerus of each side was X-rayed. The right showed an oblique fracture just below the middle (*Figs.* 324, 325). Both showed subperiosteal new bone

FIG. 327.—The skull.

formation which blended with the cortex ; this was irregularly rarefied and showed a trabecular structure very clearly. The eccentric thickening of the cortex and the clear delineation of its texture were somewhat reminiscent of the appearances in Paget's disease.

A screen examination of the whole patient was carried out, and radiographs were made from those bones which appeared affected when so viewed. These comprised all the bones of the body except the pelvis, skull, hands, and feet. The changes were similar to those described above, except that localized patches of decalcification were more pronounced in some situations—left radius and ulna (*Fig.* 326), both fibulæ.

PROVISIONAL DIAGNOSIS.—Syphilis was considered the most likely cause of the generalized bone condition associated with the pathological fracture, although the patient was old to be giving the first indications of congenital disease, of which stigmata were absent, and seemed young for the bony manifestations of acquired disease. The most probable differential

diagnosis was considered to be generalized fibrosis of bone (' osteitis fibrosa ' without hyperparathyroidism), or possibly hyperparathyroidism.

FURTHER INVESTIGATIONS.—

Urine.—Bence-Jones albumose was absent.

Blood Wassermann Reaction.—This was negative on three occasions, the last after a provocative dose of N.A.B. (0·45 g.).

Further X-ray Examination.—Radiographs were made from those parts of the skeleton which had been examined previously on the screen only (e.g., *Fig.* 327). All showed the changes described, and they were particularly beautifully seen in the hands and feet.

Blood Chemistry (Dr. H. E. Archer, Dr. G. A. Harrison, and Dr. William Smith).—

Serum calcium—

13·0 mg. per 100 c.c. serum (1st exam.).

11·3 mg. per 100 c.c. serum (2nd exam.).

Inorganic phosphorus—

3·7 mg. per 100 c.c. plasma (1st exam.).

3·4 mg. per 100 c.c. plasma (2nd exam.).

Plasma phosphatase 0·788 units.

FINAL DIAGNOSIS.—Although nothing abnormal had been reported in the screen examination of the pelvis, the radiograph clearly showed the tracks of intramuscular injections. When confronted with these the patient admitted to having received treatment for acquired syphilis at another hospital.* He had not previously admitted to any illnesses, but had not been very closely questioned. In this connection it is worth recording that he was prosecuting a claim for compensation for his accident against the tramway undertaking.

PROGRESS of FRACTURE.—A Robert Jones humerus extension splint was applied. At the end of four weeks clinical examination revealed union, and active movements of shoulder and elbow were started. Full function was regained.

PROGRESS OF GENERAL OSSEOUS CONDITION.—Seventeen months after the first examination fresh skiagrams were taken of the bones of each arm (*Fig.* 328), forearm, hand, thigh, and leg. They had returned to an almost normal condition.

The patient stated that he had not received any further treatment.

FIG. 328.—The right humerus after an interval of seventeen months from the date of *Fig.* 325.

COMMENTS

The features of particular interest in this case are the following :—

1. The onset of generalized osseous changes in spite of intensive treatment, which had started in the primary stage and was still being carried out when the pathological fracture occurred, and which had resulted in a negative Wassermann reaction.

2. The secondary character of the condition, as shown by its early onset, by its generalized distribution throughout the skeleton, and, if the patient's statement is accepted, by its spontaneous resolution.

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* I am much indebted to Dr. V. E. Lloyd for the following information :—

Fifteen months previously, first attendance with primary penile lesion (*Sp. pallida* present) and inguinal adenitis. Wassermann ++. No secondary signs. Treatment with arsenobenzole and bismuth injections. Wassermann negative prior to second course of treatment, starting six months after the first. Wassermann negative also at end of third course of treatment fourteen months after the onset. Total medication in fourteen months : N.A.B. 12·85 g.; bismuth oxychloride, twenty-seven injections of 2 c.c.

3. The complication of a pathological fracture, and the readiness with which this united.

4. The circuitous method of arriving at a diagnosis.

5. The medico-legal implications.

6. The possibility that the generalized bone changes were the result of therapy and not the result of the disease.

In addition to the acknowledgements made in the text I have to thank Mr. R. C. Elmslie for taking the patient under his care for certain of the investigations; and Dr. J. Wylie, Dr. D. M. Stone, and Dr. G. T. Loughborough for permission to reproduce the skiagrams.

A COMPARISON OF THE RESULTS OF SPINAL FIXATION OPERATIONS AND NON-OPERATIVE TREATMENT IN POTT'S DISEASE IN ADULTS

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EVIDENCE, both clinical and pathological, is in favour of the view that the tubercle bacillus in a 'cured' lesion is not destroyed by the body; it is only encapsulated by the reactive changes of the host and is thereby rendered more or less innocuous. This conception of the disease is probably not invariably true, but for practical purposes it must be held that a tuberculous focus always carries the danger of containing live tubercle bacilli.

In order, therefore, to obtain a permanent cure of the disease, excision should be very satisfactory; but actually its application is limited because, firstly, general treatment is always required as well as treatment for the local lesion; secondly, excision is frequently not complete; and thirdly, some lesions are very difficult of access. In early tuberculous lesions of bone before joint involvement has occurred, excision of the focus is often advocated, and is probably a sound line of treatment; but in the case of the spine, with a few heroic exceptions, it is never attempted. Consequently, until some specific remedy is introduced, we are left with 'cure' by conservative treatment, which means encapsulation of the focus by fibrous tissue. This is a fairly satisfactory result except under two conditions, one where a communication with a blood-vessel persists and metastatic lesions occur repeatedly, and the other where movements take place (i.e., in joints) which are liable to cause liberation of the bacilli and reactivity of the disease. The former accident is as a rule beyond our control, but the latter can be obviated to some extent by treatment. It is this decrease in the danger of reactivity that has, generally speaking, caused the absolutely fixed joint to become recognized as the best end-result of tuberculous disease.

As the absolutely fixed joint (true bony ankylosis) is very seldom achieved in cases of pure tuberculous infection, artificial fixation of the joint was suggested, and at present plays a big part in the treatment of the condition. The problem of the stage at which the operation should be performed is one which requires very careful consideration, and the main object of this article is to help to establish the place of fixation operations in spinal disease in adults.

The idea of absolute immobilization afforded by operative aid, the short-cutting of the natural healing process, the essentially conservative nature of the operation, its relatively easy technique and low mortality-rate, all combined to render treatment by the creation of bony ankylosis very popular, especially in cases where the economic factor had to be considered. The lack of anterior consolidation induced by early fixation and the increased frequency of late abscess-formation were not appreciated until later.

The literature on this subject is (as is the way with all controversial subjects) relatively enormous, there being more than forty articles published during the last three years. A perusal of this and previous literature is useful in that it shows the cautious attitude adopted by the leading authorities on spinal tuberculosis in England, in contrast to the enthusiasm with which the operation was advocated in America.

Albee[1] first performed his operation in 1909 and Hibbs[2] in 1911. Later, in 1918, Hibbs reported a series of 210 cases without any primary mortality and with only 4 cases in which pseudo-arthrosis formed subsequently. This was a very satisfactory report as far as immediate results were concerned, and in view of this as well as of the other appealing factors in favour of the operation, the American enthusiasm was sustained for many years, and even as late as 1935 the operation was advised on any patient at any age, provided the surgical risk was good. That there were limits to the indications for the operation, however, has been expressed on many occasions, and the general idea of these may be obtained from the following quotations from the literature.

In 1923 Girdlestone,[3] concluding an analysis of 100 cases, stated: " In adults posterior spinal fixation is reliable and has great value as part of the conservative treatment of Pott's disease." Gauvain,[4] in 1933, is of the opinion that " in adults their employment is more often justifiable, but I still hold the view that they are not indicated when the disease is active, and that, in other words, their value is rather in the nature of effective internal splintage to replace ineffective external splintage ". King,[5] in 1933, reporting on the results of 93 cases that had had spine fusion operations, makes these interesting observations: " The stability of the kyphos is mainly dependent upon adequate anterior vertebral-body contact. Correction obtained by abduction of its jaws causes instability by producing a dead space in the diseased area. Posterior spinal fusion does not restore sufficient stability to maintain correction obtained in this manner ". Calvé,[6] in 1935, advocated that it should only be performed when the disease is in the stage of an end-result, and this may be taken as a summary of the present-day opinion of ' when to operate '.

The series of cases analysed below supports this view in general, and not only confirms the fact that the stability of a kyphos is mainly dependent on adequate anterior vertebral-body contact, but also the results indicate that posterior fixation by operation in the early stage of the disease delays anterior fixation between the vertebral bodies (apparently because it prevents approximation in the later stages of the healing process), and, as a result of this lack of consolidation, abscess formation and reactivity are more common in the cases treated by operation.

CHOICE OF CASES FOR COMPARISON

A comparison of the results of 100 cases treated by non-operative means and 50 cases treated by fixation operations has been made, together with an attempt to obtain cases of the greatest comparative value. For this purpose those with the following factors in common have been chosen, but beyond this there has been no selection of cases.

Age.—All cases were of sixteen years of age or older at the commencement of the disease.

Site.—All the lesions were in the dorsal or lumbar regions.

Stage of Evolution of the Disease.—All the cases were in an early stage of the *initial* activity at the commencement of the treatment, so that cases with a hopeless prognosis on admission because of old-standing disease, incorrect previous treatment, or lack of treatment altogether are not included.

General Treatment.—All cases have had the same general treatment at the same sanatorium, but whereas in those treated by operation it has been for twelve months following the fixation, in those without operation it has been continued for a period of eighteen months on the average.

CLASSIFICATION OF CASES

The comparative value of the cases is still further increased by a radiological classification of the initial lesion, a radiological classification of the end-result, a clinical classification of the end-result, and an efficient follow-up scheme. The initial radiological classification shows the extent of the disease in the two groups of cases at the commencement of treatment, and indicates that on the whole a rather more extensive type of lesion has been dealt with by operation, but the differences are not sufficient to prevent a reasonable comparison, as given in *Table I.*

Table I.—Extent of Disease in Operation and Non-operation Cases at Commencement of Treatment (Initial Radiological Classification)

Class of Lesion	Operation Cases	Non-operation Cases
	per cent	per cent
1. Minimal lesion..	8	17
2. Metaphysial and osteomyelitic of two vertebral bodies	66	73
3. Similar to (2) but involving more than two vertebral bodies ..	26	10

Classification A: The Radiological Appearance of the Initial Lesion.—It is convenient to consider this on a basis of the degree of bony destruction. Three main classes may thus be formed :—

Class 1.—A minimal lesion, usually metaphysial[7] (often called tuberculous epiphysitis), sometimes periosteal, or even both metaphysial and periosteal (*Fig.* 329).

Class 2.—A metaphysial and osteomyelitic lesion involving not more than two adjacent vertebral bodies.

Class 3.—Similar to *Class* 2 but involving more than two vertebral bodies.

Classification B: The Radiological Appearance of the End-result.—This classification is made on a basis of the degree of fusion anteriorly between the vertebral bodies as seen on the X-ray films. The lesions must also show good surrounding density with a clear outline, thus indicating quiescence.

Class 1.—Definite complete fusion of the diseased area. The majority of these will probably be bony ankylosis (*Fig.* 330).

Class 2.—A minimal degree of incomplete fusion as evidenced by a linear gap at the site of the lesion. This type will correspond to firm fibrous ankylosis pathologically.

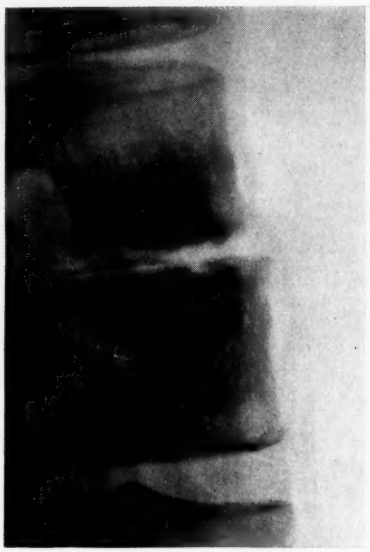

FIG. 329.—A *Class* 1 initial lesion—i.e., minimal degree of involvement.

FIG. 330.—A *Class* 1 result, same case as *Fig.* 329.

FIG. 331.—A posterior dislocation. This type of end-result is only seen in cases which have an onset in childhood.

Class 3.—A very definite gap (a quarter to half an inch) between the bones affected at the site of the lesion. This will correspond to insecure fibrous union pathologically.

Class 4.—An extensive loss of substance at the site of the lesion, giving rise to either a gap greater than the depth of one whole vertebral body or to a pathological dislocation.

It must be realized that some cases which started as minimal lesions may remain so localized that complete destruction of the cartilage does not occur. In these cases a small gap will be seen in the radiograph; this will not represent an area of cavitation, however, but merely the remains of an intervertebral disc. When *Class* 1 and *Class* 2 radiological end-results are considered together, as GOOD, as in *Table II* and as used in the comparison of the cases, an error due to the misinterpretation of the radiographs is avoided.

Classification C: The Clinical Condition of the End-result.—

Class A.—Up and about, fit for full day, no symptoms.

Class B.—Alive and well, but only up part time, minor symptoms.

Class C.—Reactivity of the spinal disease, or the occurrence of any complication such as abscess formation, sinus formation, and paraplegia.

Class D.—Died.

The degree of deformity (kyphos) has not been considered in classifying the end-results of these cases. This is due to the fact that, firstly, the cases with the soundest fusion often show well-marked deformity; secondly, the gross deformities are seldom seen in cases which have not had an onset in childhood (*Fig.* 331), and so the question of degree of deformity is of minor significance in the adult, even from a cosmetic point of view.

In *Table III* only *Class A* clinical results are considered GOOD.

The Follow-up Scheme.—Under the London County Council all cases of a tuberculous nature are followed up for a minimum period of five years after they have returned to home conditions and provided that they remain quiescent. They are also as a rule supervised by the same medical officer who has had charge of them during their stay in hospital.

The length of time in which the cases comprising the clinical groups *A* and *B* have been under observation as quiescent and under home conditions is an average of five years for the operation group and three years for the non-operation group.

COMPARISON OF END-RESULTS

(*Tables II, III, IV*)

The end-results, in order to simplify comparison, may be summarized as follows:—

Non-operative Treatment.—Gives:—

a. 62 per cent GOOD radiological results—i.e., cases showing bony fusion or fibrous fusion with a minimal gap.

b. 70 per cent GOOD clinical results—i.e., cases which are back under home conditions, are fit for a full day of work, and have no symptoms.

c. 4 per cent of cases showing late abscess formation, i.e., after they had finished treatment and had been discharged.

Operative Treatment.—Gives:—

a. 38 per cent GOOD radiological results.

b. 32 per cent GOOD clinical results.

c. 40 per cent of cases showing late abscess formation.

These main results of the analysis are so striking and decisive that, even allowing for the slightly more extensive type of lesion dealt with and the less general treatment received by the operative group, the following definite conclusions can be formed :—

1. Cases treated by non-operative means give better anterior consolidation.
2. Cases treated by non-operative means give better clinical results.
3. Cases treated by non-operative means are much less liable to develop late abscess formation.

Table II.—Comparison of End-results Radiologically

Class of End-result		Operation Cases	Non-operation Cases
1. Bony ankylosis	Good	per cent 8	per cent 10
2. Firm fibrous ankylosis	Good	30	52
3. Insecure fibrous ankylosis ..		46	36
4. Large gap or dislocation ..		4	—
5. Remained active until death ..		12	2

Table III.—Comparison of End-results Clinically

Clinical Class	Operation Cases	Non-operation Cases
A. Normal life. Good ..	per cent 32	per cent 70
B. Restricted life	14	6
C. Reactivity	32	14
D. Died	22	10

Table IV.—Chief Complications and Time of their Occurrence in History of Disease

Complication	Operation Cases			Non-operation Cases		
	Before Operation	During After-treatment	After Discharge	Before Treatment	During Treatment	After Discharge
Abscess ..	per cent 26	per cent 10	per cent 40	per cent 54	per cent 1	per cent 4
Sinus ..	4	—	2	8	2	1
Paraplegia ..	6	—	2	8	2	1

FURTHER RELATIONSHIPS BETWEEN DIFFERENT ASPECTS OF SPINAL DISEASE, OF VALUE IN ASSESSMENT OF TREATMENT

It seems almost axiomatic to say that the firmer the anterior fusion the better the clinical result.

Table V definitely confirms this view, but perhaps not to such an extent as would be expected; this may, however, be due to the fact that bony ankylosis seldom occurs without secondary infection, and secondary infection is very prejudicial to a good clinical result. It must be clearly understood that " firm ankylosis, preferably bony, is the thing aimed at ", as was enunciated by Percival Pott nearly 200 years ago.

The initial X-ray lesion has a very definite bearing on the X-ray end-result; this is shown by *Table VI*, in which it will be seen that in the cases treated by non-operative measures :—

Minimal lesions (*Class* 1) give 16 out of 17 results (94 per cent) of bony ankylosis or of minimal-gap fibrous ankylosis—i.e., the class of result which is considered GOOD from the radiological point of view.

Table V.—RELATION OF DEGREE OF ANTERIOR FUSION TO CLINICAL RESULT IN NON-OPERATIVE GROUP

DEGREE OF ANTERIOR FUSION (X-RAY RESULT)	CLINICAL RESULT			
	Class A	*Class B*	*Class C*	*Class D*
Bony fusion (*Class* 1) ..	per cent 80	per cent 10	per cent 10	per cent —
Fibrous union with minimal gap (*Class* 2)	71·2	3·8	15·4	9·6
Fibrous union with moderate gap (*Class* 3)	69·5	8·4	13·4	8·4

Table VI.—RELATION OF RADIOLOGICAL LESION AT COMMENCEMENT OF TREATMENT TO RADIOLOGICAL END-RESULT IN NON-OPERATIVE GROUP

INITIAL LESION	END-RESULT				
	Class 1	*Class* 2	*Class* 3	*Class* 4	Remained Active
Minimal lesions (*Class* 1) ..	3 cases	13 cases	1 case	—	—
Involving two vertebral bodies (*Class* 2)	7 cases	36 cases	28 cases	—	2 cases
Involving more than two vertebral bodies (*Class* 3) ..	—	3 cases	7 cases	—	—

Lesions involving the bodies of only two vertebræ (*Class* 2) give 43 out of 73 cases (58·9 per cent) of GOOD results radiologically.

Lesions involving more than two vertebral bodies (*Class* 3) give only 3 out of 7 cases (43 per cent) of GOOD results.

A further significant point is that there are no *Class* 4 end-results in the non-operative group (100 cases); the tendency of the disease to remain localized if correct treatment is instituted is thus well shown. When the onset of the disease is in childhood, however, *Class* 4 end-results are relatively common (cases with gross degrees of deformity), in marked contrast to the adult form.

CONSERVATIVE TREATMENT

The length of time required by conservative treatment in adults is actually remarkably constant, the average being eighteen months, again a marked contrast to the more prolonged and highly variable period of treatment required when the onset is in childhood.

The method of treatment used is based on the pathological evolution of the disease as demonstrated by serial radiography and occasional pathological specimens. It may be summarized as follows :—

1. *To Obtain Quiescence.*—For this purpose immobilization in recumbency is required until the activity as demonstrated clinically and radiologically has subsided. A spinal frame or plaster-of-Paris bed is the method employed, and about twelve months is the period of time required. A moderate degree of hyperextension is used at this stage, just sufficient to create a state of absence of compression in the diseased area, and so mechanical destruction as far as possible is prevented.

2. *To Obtain Consolidation of the Diseased Area, when Activity has Subsided.*—This is carried out by firstly allowing the patient to be free in bed for about two months, and then allowing him to get up in a spinal brace, taking about three months before the full normal activities are resumed.

The degree of kyphosis is so slight, as a rule, in adults, that the production of secondary curves by extension above and below the diseased area is not required.

ILLUSTRATIVE CASES

The following case reports are of interest as illustrations in themselves of the main contentions of this article.

Case 1.—W. A. D., male, aged 22 years.

This case showed two lesions. The initial one of the eleventh and twelfth dorsal vertebræ was fixed by a graft extending from the spinous process of the tenth dorsal vertebræ to that of the first lumbar, the operation having been performed five months after the onset of symptoms. Post-operative treatment consisted of two months in a plaster-of-Paris bed; the patient was then allowed up wearing a spinal brace, and one month later commenced work as a saw-mill hand. This work was continued for two years, when the pain in the back recurred and radiological examination showed a second lesion between the eighth and ninth dorsal vertebræ.

The full conservative treatment was carried out for this second lesion, and the final X-ray film is very interesting. It is reproduced diagrammatically in *Fig.* 332, and shows that there is a definite gap (*Class* 3 result) at the site of the initial lesion, but that the second lesion has consolidated well, no cavity remaining (*Class* 2 result). On further examination of this film a slight degree of erosion of the anterior surface

of the tenth dorsal vertebra can be made out, and a faint shadow anterior to the affected region of the spinal column, suggestive of an abscess between the anterior common ligament and the vertebral body (aneurysm phenomenon). This probably indicates that excessive movement and strain at the site of the eighth and ninth dorsal vertebræ, as a result of the fixation of the spine below, were the factors that determined the level of the second lesion.

Case 2.—M. M., aged 37 years, female.

This case was a metaphysial and osteomyelitic lesion of the tenth and eleventh dorsal vertebræ, i.e., *Class* 2. It was grafted, while still active, about six months after the onset of the disease, the graft extending from the spinous process of the

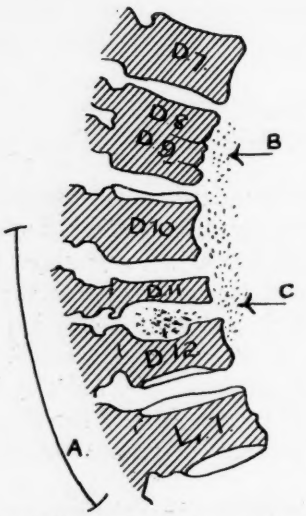

FIG. 332.—*Case* 1.—End-results of operative and non-operative treatment compared in the same case. Both show quiescence of the disease, but whereas there is good consolidation in the non-operative instance, a cavity is still present in the grafted lesion. A, Extent of graft—spine of D.10 to that of L.1: B, Second lesion, good density, clear outline *and good consolidation—Class* 2 end-result; C, First lesion, good density, clear outline *but cavity still present—Class* 3 end-result.

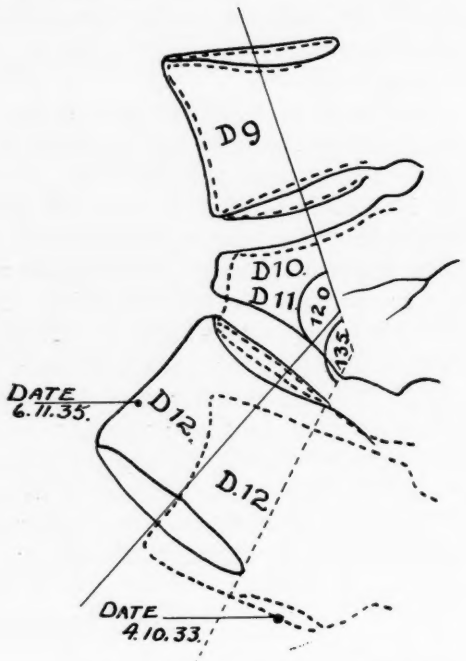

FIG. 333.—*Case* 2. Superimposed tracings of X-ray films showing 15° of collapse as a result of poor anterior consolidation, in spite of a graft.

eighth dorsal to that of the twelfth dorsal vertebra. After-treatment was prolonged, the total period of recumbency being fifteen months, because of abscess formation and paraplegia which arose six months after the operation. A comparison of the X-ray film immediately after grafting and the X-ray on discharge over two years later (*Fig.* 333) shows that further collapse of the diseased area has taken place.

This case illustrates the fact that an early graft will hold the diseased area sufficiently to prevent consolidation and yet will not be sufficiently strong as a sole means of support to prevent minor degrees of collapse from taking place.

Case 3.—G. A., aged 28 years, female.

This lesion of the twelfth dorsal and first lumbar vertebræ, of the metaphysial class, was grafted during the active stage of the disease and followed by full conservative treatment, giving a *Class* 1 X-ray result (bony fusion). A few months after

discharge the patient felt something snap in her back, and a radiograph (*Fig.* 334) taken at this time revealed a fracture of the graft.

The following points are of special interest in this case :—

1. The graft was placed too high so that it did not span the diseased area (this occurred in one other case in the series of 50).

2. Full conservative treatment gave a *Class* 1 result.

3. The strain on the portion of the spine unnecessarily fixed by the graft was sufficient to fracture the graft.

Case 4.—W. B., female, aged 36 years.

This case was grafted during the active stage of the disease, the graft extending from the spinous process of the eighth to that of the twelfth dorsal vertebra, the

FIG. 334.—*Case* 3. A *Class* 1 result. The graft spans the wrong spines, and also shows a fracture in its centre.

FIG. 335.—*Case* 4. This shows extensive involvement of the lower dorsal region. The lowest lesion has a sequestrum in its centre, and is held open by a graft, while the upper lesion shows good consolidation.

lesion being a metaphysial and osteomyelitic one (*Class* 2) of the ninth and tenth dorsal vertebræ. After-treatment consisted of fourteen weeks' recumbency followed by a plaster-of-Paris jacket for four months, and then the patient was fitted with a spinal brace and recommenced her work as a machinist (sitting occupation).

After fifteen months under working conditions root-pains occurred, and the X rays showed a recurrence both above and below the original lesion. Further conservative treatment has now been employed for eighteen months, and the latest X-ray film (*Fig.* 335) shows that the extension of the disease was probably by way of the anterior abscess, the cupping of the anterior outline of the vertebral bodies being quite definite (aneurysm phenomenon), and the shadow representing the abscess anterior to the column fairly distinct. The lower lesion between the eleventh and twelfth dorsal vertebra is bridged by a graft and shows a wide cavity with sequestrum formation. The original lesion of the ninth and tenth dorsal

vertebræ shows fairly good consolidation. The upper lesion between the seventh and eighth dorsal vertebræ does not show the cavity formation like the lower one, because it has not been bridged and consequently held open by the graft.

PROGNOSIS

The prognosis in Pott's disease in adults is often quoted as being very grave, but this is not the case if correct conservative treatment is employed. *Table III* shows a mortality-rate of 10 per cent for 100 cases which had been under observation for an average period of six years since the onset of the disease. Actually the prognosis as regards life, length of treatment required, and end-result is much better in adults than in children.

A CONSIDERATION OF CERTAIN POINTS OFTEN PUT FORWARD IN FAVOUR OF OPERATION

The Economic Factor.—It is because of the economic factor in adults that operative fixation is so often advocated, but it is now agreed that general treatment for at least a year is required in all cases, and as conservative treatment only takes eighteen months the actual saving of time is only six months. But at what sacrifice is this six months saved? When it is realized that with operation there are only 32 per cent GOOD results, whereas with conservative treatment there are 70 per cent GOOD results, this economic factor as an indication for early operation cannot be taken into consideration.

The Immobilization Factor.—A spinal graft, after it has 'taken', together with recumbency, is the most effective means of obtaining immobilization; in fact it is the only way whereby movements during respiration can be completely prevented. This factor is the only unequivocal advantage of early grafting.

The Vascularizing Function of the Graft.—It has been suggested that the insertion of a tibial graft causes an increase of the vascularity of the diseased area of the spine (Albee) and so has a beneficial action on the course of the disease. The centre of a tuberculous lesion has an impoverished blood-supply, but the periphery has a zone of hyperæmia; it must be extremely difficult to decide that this zone of hyperæmia had been still further increased by a graft except possibly as a result of the minute study of a very large number of post-mortem examinations. However, allowing for such a possibility, the end-results of these cases suggest that there has either been no such beneficial influence, or, if there has, then it has been more than counteracted by the disadvantages of grafting during the early stage of the disease.

THE PLACE OF SPINAL FIXATION OPERATIONS IN THE TREATMENT OF POTT'S DISEASE

1. During the Active Stage.—As a result of this analysis it may be concluded that the operation of spinal fixation is contra-indicated in the active stage of Pott's disease in the adult, because :—

a. The clinical end-results are only half as good as those obtained in conservatively treated cases.

b. Anterior fusion between the vertebral bodies is delayed by posterior spinal fixation.

c. Posterior fixation is not sufficient to stabilize the spinal column, if there is poor anterior fusion.

d. The economic factor is put out of consideration because of the inferior end-results obtained by operation; further, if general treatment is used (and it should be used in all cases irrespective of local treatment) the saving of time is only six months.

In brief, the only object in performing the operation at all during the active stage is because of its immobilization effect, and this may be obtained sufficiently well by non-operative means.

2. In the Treatment of the End-result.—Spinal fusion used solely as a form of internal spinal support for the diseased area, when the optimum degree of anterior consolidation has been obtained, is much more efficient and much less cumbersome than any form of external appliance, and undoubtedly has a most useful place in establishing the permanent cure of the disease. A consideration of the radiological end-results obtained in this disease brings out the conclusions that :—

a. In the cases with bony ankylosis (*Class* 1 type), where fusion is sound anteriorly, then probably nothing further is required; in fact, putting a graft into such a case is unnecessary (though it would be wiser to graft in a case where there was a doubt as to whether bony ankylosis had occurred or not).

b. Cases of fibrous union (*Classes* 2, 3, 4) are definite indications for grafting, but with the provision that in the case of a large loss of substance—i.e., *Class* 4—it must be a true end-result and must have been given many months, even years, to consolidate as much as possible anteriorly before the operation is carried out.

THE TYPE OF FIXATION OPERATION

There are several satisfactory methods of performing spinal fixation. An autogenous tibial graft combined with obliteration of the posterior articulations is probably the best, but what is most important is that it should immobilize the diseased area and the diseased area only. The reasons for advocating this short type of graft may be enumerated as follows :—

1. If the graft includes the spinous processes, posterior articulations, and laminæ of the diseased vertebræ, then the actual area of disease will be immobilized efficiently—i.e., it is efficient as a form of spinal support for the weakened region.

2. When the disease has reached the stage of an end-result, there can be no object in throwing out of action normal intervertebral joints above and below the lesion. In fact, there is a positive objection to such a procedure, because the greater the length of spine immobilized the greater the stresses and strains on the rest of the column, if normal activities are undertaken, giving an increased liability to recurrence above and below the diseased area.

SUMMARY

The treatment of tuberculosis of the spine is discussed and the literature on fixation operations briefly reviewed.

A series of 100 cases treated by non-operative means and a series of 50 cases treated by operation are analysed and compared, chiefly in regard to their end-results.

The main conclusion arrived at is that spinal fixation operations performed during the active stage of Pott's disease prevent consolidation between the vertebral bodies at a later stage in the healing process; and that consequently the place of operative aid is solely in the treatment of the end-result, in order to establish a permanent cure of the condition.

I am indebted to Dr. Colvin, of St. Luke's Hospital, Lowestoft, for suggesting the investigation.

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GASTROSCOPY

ITS HISTORY, TECHNIQUE, AND CLINICAL VALUE, WITH REPORT ON SIXTY CASES

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HISTORY

The first attempt to view the interior of the stomach without having recourse to abdominal section was made by Kussmaul in 1868. The incandescent electric lamp had not at that time been invented, and Kussmaul's efforts were made by the rays of light from a naked flame reflected down a long straight wide tube. The clinical results were of no practical value at the time, but the attempt was an expression of the growing desire of clinicians to render visible the internal as well as the external surfaces of the body.

In 1871, Desormeaux laid down the general principles of endoscopy, and in 1876, just sixty years ago, the first indirect vision gastroscope was made by Nitze in Vienna. This instrument had an optical system similar to that of a modern cystoscope, with an electrically-heated incandescent platinum wire as the source of illumination. Difficulties were encountered with the very large diameter of the tube and the heat emitted by the platinum wire, and this instrument was only used experimentally on the cadaver. Mikulicz, however, interested himself in the problem, and in 1881 the first practical gastroscope was made under his direction. This instrument was narrower, and the glass enclosing the incandescent platinum wire was kept cool by a stream of water. In order to clear the projecting vertebral column the instrument was angulated through 30° near its distal end, with a prism at the angle to deflect the light rays to the eye-piece. Several patients were successfully examined with this instrument by Mikulicz with no fatalities, and in one case a carcinoma of the stomach was diagnosed by means of it. General anæsthesia was necessary for the instrumentation, which was described by Mikulicz as "an exquisite surgical manipulation which should only be undertaken by the most expert". Mikulicz, however, was disappointed with his experience. The view of the stomach was incomplete on account of the lack of a rotating nose-piece to the angulated instrument, so that a negative result from the examination was of no significance. In consideration of this and the necessity for general anæsthesia, Mikulicz held that exploratory laparotomy was to be preferred, and abandoned the method.

The effect of the practical failure of this very brilliant piece of work was to render clinicians sceptical of the possibilities of gastroscopy for the next thirty years. A number of unsuccessful attempts to solve the difficulties were made during this period. Rosenheim experimented with a straight rigid instrument, but found it too dangerous to use. Rewidzoff tried to make this safer by first introducing a flexible stomach tube and passing the rigid instrument through this protecting

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channel. It was found, however, that with a straight gastroscope the projecting lumbar vertebral column limited the intra-gastric field of vision considerably, and the results obtained were considered by the majority of practitioners insufficient to justify the discomfort entailed in the use of this rigid type of instrument.

In 1900 Kelling devised an instrument consisting of a number of hinge-jointed sections so that it could be introduced as a relatively flexible tube, but braced straight when in position by longitudinal tension wires. This instrument was angulated like that of Mikulicz to clear the vertebral column, but it also had the rotating nose-piece that Mikulicz had desired so much in order to see the whole area of the stomach. It was unfortunately too complicated and fragile, and was soon irreparably damaged before its use could be proved.

The solution of the problem was carried a stage further in this country by H. S. Souttar and Theodore Thompson in 1908. The experience of past workers together with a wide knowledge of physics and a remarkable degree of mechanical ingenuity enabled Souttar to construct an instrument which fulfilled all the physical requirements. It was angulated, carried a rotating nose-piece, and was constructed on the sectional principle of Kelling, so that it could be passed flexible and braced rigid for the examination. Souttar and Thompson successfully examined a number of cases at the London Hospital, which they reported in the *Quarterly Journal of Medicine*. The use of this instrument, however, was allowed to lapse for two reasons. In the first place, although a good view was obtained, general anæsthesia was necessary for the instrumentation, and so many cases subsequently had an operation that the anæsthetic for the gastroscopy was felt to be hardly justifiable. Also, radiological examination of the stomach by the barium meal, which was at that time coming into general use, seemed to satisfy all the requirements of gastric investigation. For the second time, therefore, gastroscopy was abandoned after it had been successfully practised.

As the limitations of radiology of the stomach became gradually recognized on the Continent, however, clinicians there turned again towards the ideal of gastroscopy and the problem of how to render it safe, reliable, and painless. The solution was found in a new optical system, in which a flexible telescope was made capable of transmitting an image notwithstanding a moderate degree of curvature of the tube. This was accomplished by the inclusion of a large number of lenses, each of which projected an image of small dimensions relative to the diameter of the lens. In 1932, under the direction of Schindler, a flexible gastroscope was made on this principle by Wolf, of Berlin, and this instrument has altered the whole aspect of the clinical value of endoscopic examination of the stomach. Schindler, and later Henning, of Leipzig, have shown that gastroscopy with the flexible instrument is absolutely safe, and that general anæsthesia is quite unnecessary for its satisfactory performance. With these two main difficulties removed, Henning has been able to show, as the result of an experience of over 2000 instrumentations, that gastroscopy is an examination of the greatest clinical value in the investigation of gastric cases.

THE INSTRUMENT

The instrument itself consists of a rigid upper part carrying the eye-piece, and a flexible lower part carrying the objective and the electric lamp. A sponge-rubber tip beyond the lamp facilitates the safe passage of the instrument and wipes the

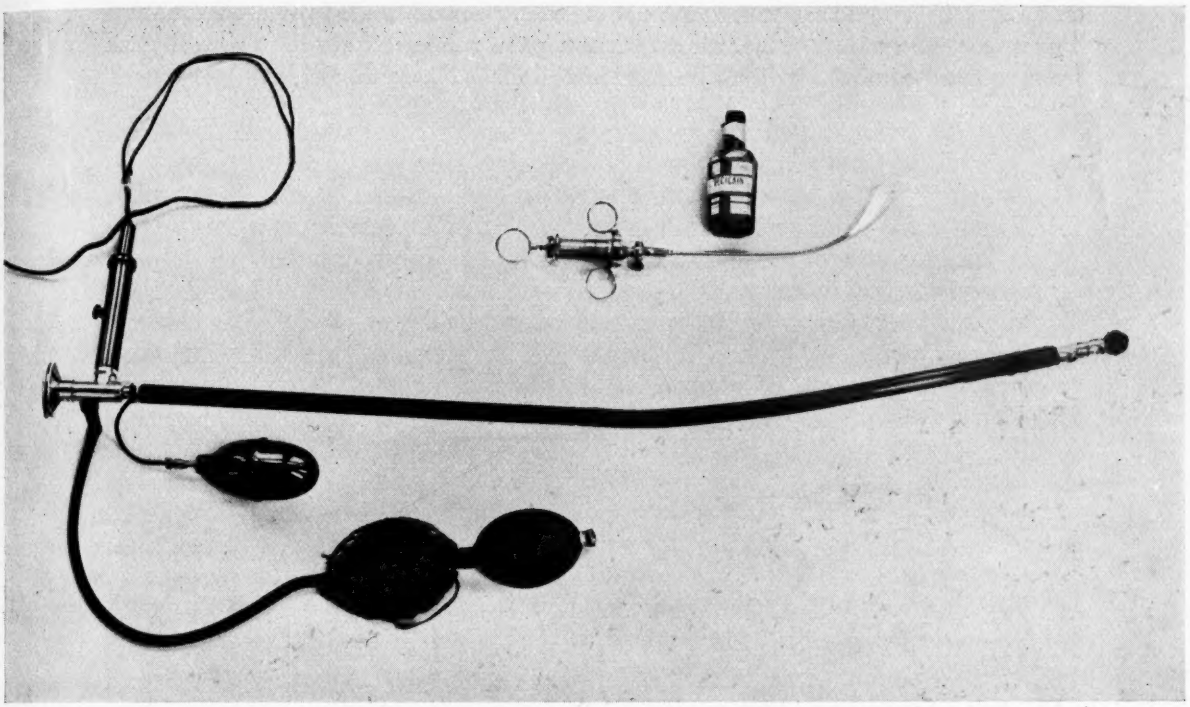

FIG. 336.—The Wolf-Schindler flexible gastroscope, with special sheath carrying the retracting balloon and inflating bulb. *Above*: The Brüning syringe for application of surface anæsthetic to the pharynx.

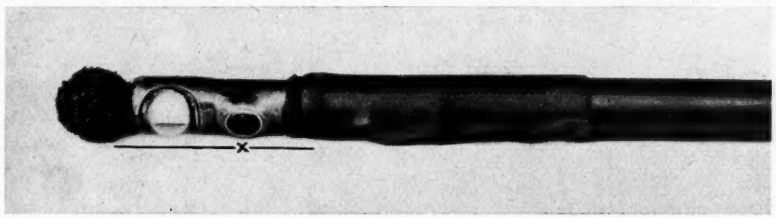

FIG. 337.—Distal end of instrument, showing sponge-rubber tip, lamp, window of objective, and balloon before inflation.

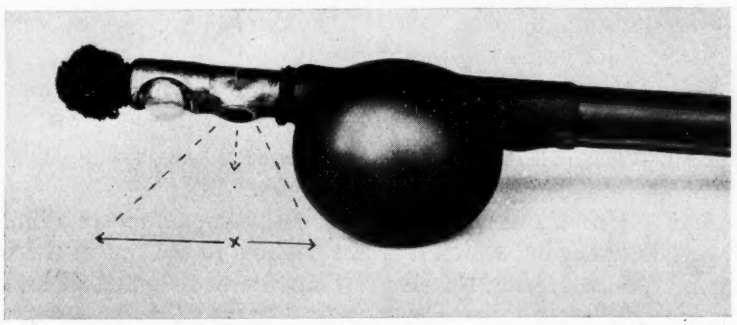

FIG. 338.—The same, showing retracting balloon inflated and lifting the objective away from the object (X) on the surface.

œsophagus in its descent, so that the optical window is not fouled by foreign matter. The upper end carries the electric connection and a rubber blower bulb whereby the stomach can be inflated with air during inspection. (*Figs.* 336, 337, 338.)

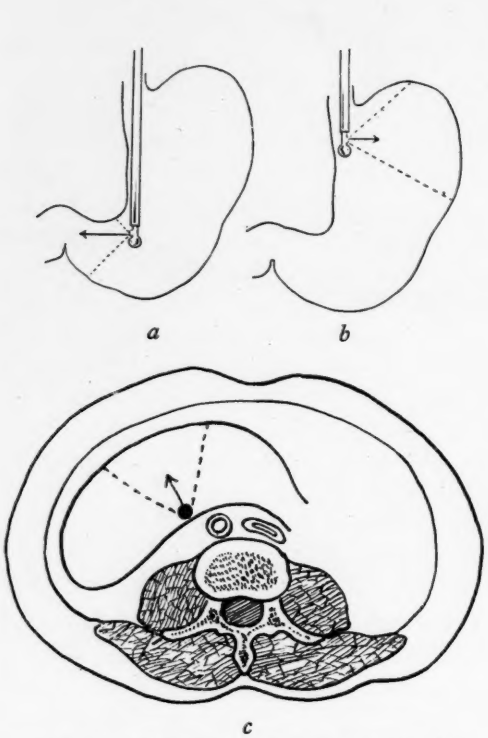

FIG. 339.—Diagrams showing gastroscopic views ordinarily obtained. *a*, Pylorus; *b*, Corpus; *c*, Anterior wall.

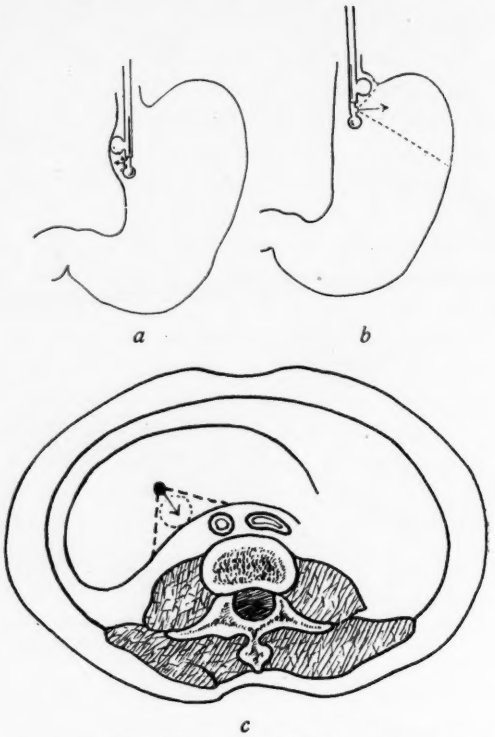

FIG. 340.—Diagrams showing views obtained with the aid of the balloon. *a*, Lesser curve; *b*, Fundus; *c*, Posterior wall.

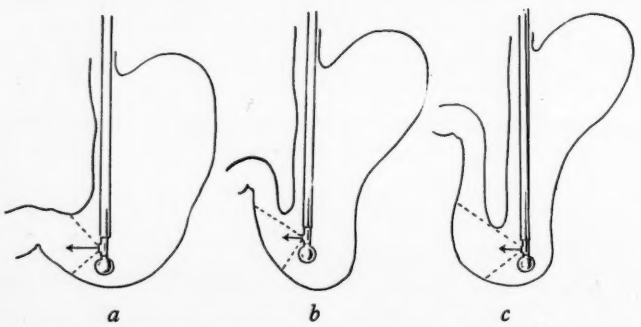

FIG. 341.—Variations in shape of stomach. *a*, Average; *b*, J-shaped; *c*, U-shaped.

The optical system of the Wolf-Schindler gastroscope imposes a limit of curvature of 34° deviation, beyond which it is not possible to see out of the objective of the instrument. Moreover, as this limit is approached, spherical and chromatic aberration render the view somewhat blurred. In practice, however, the gastroscope when in position is almost if not quite straight, and the view obtained is as sharply defined as that of the bladder on cystoscopy. Extreme degrees of flexion

of the instrument are required only for its safe introduction through the œsophagus. The 90° angle of view of the objective enables large areas of the interior of the stomach to be inspected in any one field, depending on the distance of the mucosa from the objective, and by rotating the instrument and withdrawing or advancing it in the stomach almost all of the gastric mucosa can be examined.

The position of the gastroscope in the body is as in the diagram (*Fig.* 339), rather to the left of the midline, with the objective in the corpus ventriculi. It will be seen that whereas the pyloric antrum and the greater curve of the body of the stomach lie well away from the objective and can be seen in true perspective, the upper part of the lesser curve and posterior wall of the stomach near the lesser curve are held close to the objective by the lumbar vertebral column and their proper examination presents difficulty on this account. This difficulty has been largely overcome by a modification devised by H. W. Rodgers and the author concurrently and made for us by the Genito-Urinary Manufacturing Co. This consists of a special rubber sheath designed to replace the standard sheath (*Figs.* 337, 338). It carries an inflatable rubber balloon, situated just proximal to the window of the objective and connected by a capillary rubber tube to a bulb at the upper end of the instrument. When the balloon is inflated by squeezing the bulb, it lifts the gastric mucosa out of contact of the objective and enables it to be satisfactorily inspected. *Fig.* 340 show how this device allows a proper view to be obtained of the upper part of the lesser curve, of the posterior wall near the lesser curve—the site of the stoma of a posterior gastro-enterostomy—and of the lower end of the œsophagus, while by lifting up the fundal lip of the cardiac orifice as the instrument is partly withdrawn from the stomach, a better view of the fundus can be obtained.

In a visceroptic patient the sagging of the stomach may limit the gastroscopic field considerably. Henning has pointed out that although the L-shaped stomach drawn in *Figs.* 339, 340 is that most frequently found, the viscus may be J-shaped or even U-shaped (*Fig.* 341). In the presence of a marked degree of ptosis, therefore, the dependent lesser curve may prevent a view of the pylorus from being obtained. This occurs in about 10 per cent of cases, and although it is undoubtedly a disadvantage when present, it need not necessarily prevent a satisfactory conclusion being drawn from the gastroscopy—i.e., when the suspected lesion is elsewhere than at the pylorus.

TECHNIQUE

The examination is preferably held in the morning, and except for a drink of water on waking, the patient is not allowed anything by mouth beforehand. In this way standard conditions of inspection of the gastric mucosa are obtained, when the stomach is empty and resting, and the epithelium is not stimulated to secrete by food or the thought of food.

The instrumentation can be carried out under local anæsthesia without any other pre-medication, so that it can be performed on an out-patient if necessary without undue discomfort. With a sensitive patient, however, and particularly for the first examination, it is the author's practice to give omnopon-scopolamine one and a half hours beforehand, and if necessary a second injection of morphia half an hour beforehand if the effect of the first is insufficient. It is important that the patient be not too drowsy, as his co-operation during the examination may be very helpful.

Surface anæsthesia of the mouth, pharynx, and œsophagus is obtained with 5 c.c. of 2 per cent decicaine or percaine solution to which five drops of 1–1000 adrenaline have been added. With the patient lying on his back, the mouth and pharynx are carefully painted all over with the solution—conveniently applied by a Brüning syringe (*see Fig.* 336), which keeps a wad of cotton-wool constantly wet with the liquid. It is necessary to take a full fifteen minutes over this stage, with three or four applications of anæsthetic, as complete desensitization of the pharyngeal mucosa is essential to a satisfactory examination. With proper anæsthesia, the stomach tube and gastroscope can be passed without the occurrence of any retching at all. First the lips, then the tongue, fauces, and oropharynx, are painted, and as the upper mucosa becomes insensitive, the laryngo-pharynx is anæsthetized, until on requesting the patient to swallow, the wool swab can be passed without spasm through the post-cricoid sphincter. It is important that the epiglottis and inner surface of the larynx should not be anæsthetized, as with an insensitive pharynx it may be difficult to prevent the stomach tube from entering the larynx, a mistake which cannot possibly occur if the larynx is still sensitive. It is not necessary to use a laryngeal mirror for the anæsthesia. The shape of the pharynx with the vertebræ lying posteriorly soon becomes familiar, and by keeping the swab always in contact with the posterior wall and lateral fossæ, no fluid can enter the larynx.

When fifteen minutes have elapsed since the commencement of the application of the local anæsthetic, a stomach tube is passed and the residual contents of the stomach are aspirated by a suction bottle. The character and quantity of the resting juice may here afford valuable evidence in regard to the case which may not have been present in the test-meal specimen. The patient is usually delighted to find that the stomach tube has been passed without any discomfort at all, and submits to the passage of the instrument with confidence.

Unlike cystoscopy, in which the cystoscope can be manipulated into any position inside the bladder, the gastroscope can only be rotated or moved in or out of the stomach; it cannot be moved sideways. Henning has therefore constructed a special table on which the patient can be moved, as it were, round the gastroscope, so that by tipping the patient upwards, downwards, or sideways, the stomach falls over in such a way that all parts of the interior can be brought into the field of the instrument. The author has adapted a table similar in principle to that of Henning, but he has found in practice that whereas the downward tilting of the table has often been of great use in examining the stomach, the sideways tipping of the table advocated by Henning has never enabled him to overcome a difficulty or to view an otherwise invisible area of mucosa. An ordinary operating table capable of being tilted downwards is therefore sufficient for the purpose, provided it has the necessary attachments. These consist of an adjustable head-rest, back-rest, foot-piece, and the knee-pillar invented by Henning. The author's table and the patient in position upon it are shown in *Figs.* 342, 343.

The patient lies on his left side with his back flat against the back-rest and his head moderately extended and slightly lower than the horizontal midline of the body. The upper thigh lies behind the knee-pillar and is held between it and the lower end of the back-rest, so that the patient cannot roll over. The lower knee is flexed in front of the knee-pillar and the foot is placed behind the foot-pillar, which is adjusted so that the limb is held firmly but comfortably. In this way the lower

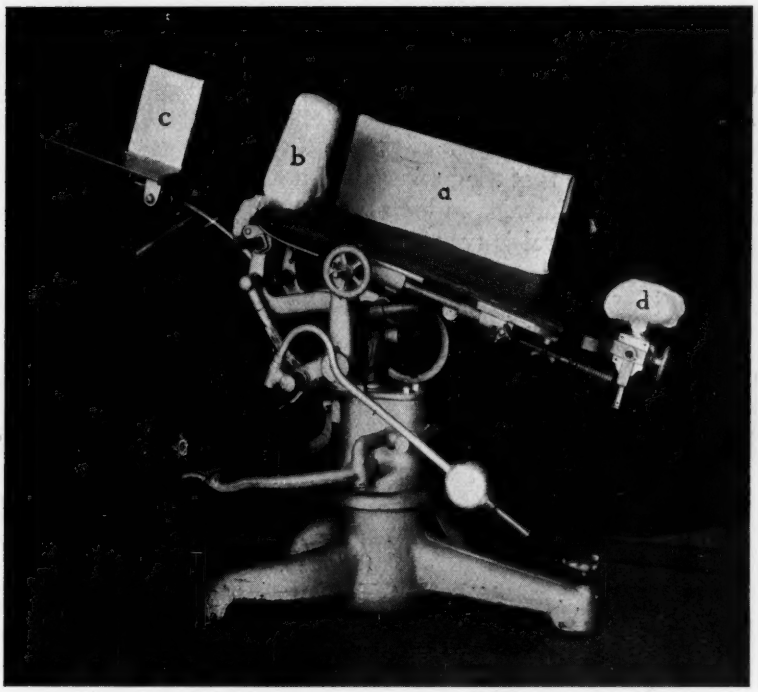

FIG. 342.—Operating table adapted for gastroscopy. Note: (*a*) Back-rest; (*b*) Henning's knee-pillar (*c*) Foot-pillar; (*d*) Adjustable head-rest.

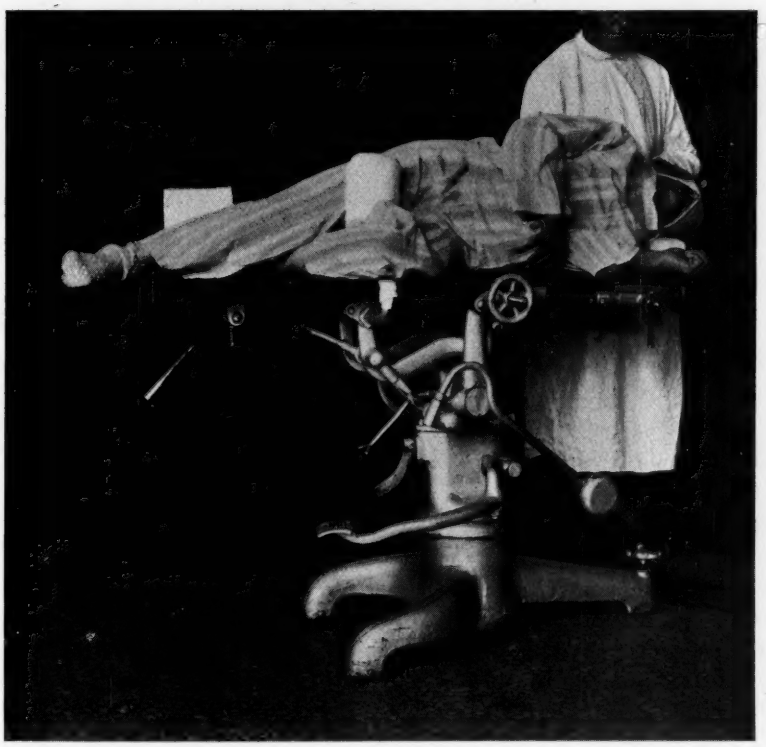

FIG. 343.—Patient in position for gastroscopy. Note the position of the lower limbs. Note also the head between the hands of the assistant.

limbs prevent the patient from slipping down the table when it is tilted, and the patient's abdomen is dependent from the pelvis and free from pressure, so that the stomach within can move freely round the gastroscope under the influence of gravity. As a further support against slipping, the patient's left shoulder is in contact with the padded lower edge of the head-rest.

It is important to see that the patient does not arch his back, as this brings the lumbar vertebral column forward in the abdomen and adds greatly to the difficulty of the examination. A towel is now placed over the patient's face and the head is comfortably supported between the two hands of the assistant. With an assistant taking the weight of the eye-piece end of the gastroscope, two fingers of the

FIG. 344.—Illustrating the grip of the end of the instrument between fore and middle fingers of left hand. The end is flexed as the instrument is introduced into the pharynx.

surgeon's left hand are passed to the back of the patient's tongue and the end of the instrument is slipped in between them, gripped, and bent forward, so that the sponge-rubber tip passes down the pharynx without pressing unduly on its posterior wall (*Fig.* 344).

As the tip impinges on the post-cricoid sphincter, the patient is asked to swallow and the instrument passes easily to the cardia. A slight delay may be experienced as the end passes through the cardiac orifice, after which the instrument glides without resistance to the bottom of the stomach.

Apart from the avoidance of loose teeth, the passage of the gastroscope presents no special difficulties if the confidence of the patient is obtained, but there is one absolute rule that must never be broken. Under no circumstances nor at any stage of the instrumentation must any force be used. The end of the gastroscope is not pushed down, but rather swallowed naturally by the patient. Gastroscopy performed in this way is not only completely painless, but quite free from danger. If any

obstruction is encountered either above or within the stomach, then this is certainly due to an organic lesion, and no attempt should be made to push the instrument beyond it by force. Such obstructions might be pharyngeal or œsophageal pouches, an S-shaped lower end of œsophagus such as might occur with a symptomless cardiospasm, or carcinoma of the upper part of the stomach, and under these circumstances (in practice extremely rare) the examination must be abandoned. With the gastroscope in position the patient should be able to lie quietly without pain or distress, beyond the discomfort caused by the displacement of the larynx to one side by the pressure of the rigid part of the instrument. He cannot of course articulate, but he can phonate, and he is able to indicate any untoward sensation to the operator.

FIG. 345.—Illustrating the position of patient, assistant, and operator during the examination. Note the instrument emerging from the right-hand side of the mouth.

Special care is taken to see that the patient's upper lip is not nipped between his teeth and the instrument, which emerges from the right side of the mouth (*Fig.* 345). The electric and pneumatic connections are then made and a small amount of air is blown into the stomach.

The first view of the interior with the objective looking downwards (i.e., to the patient's left) is the shallow pool of gastric juice—Henning's ' Schleimsee '—which lies at the most dependent part of the greater curve. The greater curve is recognized at once by the steep longitudinal folds in this region, and as the instrument is rotated forwards and backwards, the anterior and posterior walls are seen. In the normal stomach, moderately inflated, folds are present on the posterior wall, though much less prominent than the steep rugæ on the greater curve itself, but few folds exist on the anterior wall. It is advisable not to attempt at this stage to see the upper part of the lesser curve, as this needs further inflation of the stomach and

is better delayed until the end of the examination. The instrument is now advanced well into the stomach with the objective facing upwards to the patient's right. Unless the stomach is U-shaped, the whole of the pyloric antrum will now come into view (*Fig.* 346), and a little further inflation will stimulate active peristalsis. The waves of contraction can be observed passing from the mid-gastric region steadily towards the pylorus, to end in the relaxation of the sphincter and the pouting of some folds of duodenal mucosa into the orifice, sometimes with the regurgitation of a little yellowish fluid.

The whole of the pyloric region can be examined with ease, and any suspicious area can be particularly closely inspected immediately after the passage of a peristaltic wave when the mucosa of the back of the wave is turned to face the observer. The viscus is then further inflated, although never sufficiently to cause the patient discomfort, and as the instrument is slowly withdrawn the incisura angularis and the lesser curve above it come prominently into view, forming a smooth, rounded arching surface, which gets closer and closer to the objective as it approaches the cardia. The balloon is then inflated, and at once the objective can be seen to stand back from the mucosa, which can now be examined right up to the cardiac orifice.

In examining the greater curve of the stomach, it is often of great help to apply manual pressure to the abdomen, much as is done in radiological examination. The mucosa of this region can by this means be brought closer to the objective for better inspection. Difficulty in seeing the lesser curve of the pyloric antrum can often be countered by asking the patient to take a deep breath; with the cardia fixed, the descent of the liver on the right turns this part of the stomach towards the objective, so that it can be better seen in this way. Again, tipping the table steeply, so that the patient's body is inclined with the head downwards, often relieves the lesser-curve region of the weight of the liver above it in the left lateral position, so that this part of the mucosa springs farther from the objective and can be seen in better perspective. Tipping the table is also useful in moving a collection of bubbles away from an area of mucosa obscured by them. Bubbles are formed by inflating the stomach with the air orifice in contact with the mucosa, and may cause considerable or even insuperable difficulty. For this reason air must be blown into the stomach only when the end of the instrument is in the middle of the cavity and the air aperture (opposite the window of the objective) faces away from the lesser curve and posterior wall.

In examining the stomach endoscopically, notice should be taken of the general size and shape of the viscus, the degree of activity of the peristalsis, and the quantity and viscoscity of the mucus. The characters of the mucosa to be noted include the colour, presence of visible blood-vessels, areas of congestion, hæmorrhages or small erosions, and the appearance of the rugæ, whether œdematous, redundant and tortuous, or few and atrophic. Any local lesion may then be described and preferably drawn for future comparison. The examination of the stomach is not complete without a scrutiny of the minute contour of the epithelium, a magnified view of which can be obtained by examining an area of mucosa in very close apposition to the objective of the instrument. The mucosa of the posterior wall of the stomach near the lesser curve is a suitable area for this purpose, and information concerning the nature of diffuse epithelial changes can be obtained in this way.

The examination finished, the gastroscope is gently removed, and the patient is encouraged to bring up the air that has inflated the stomach. Inquiry will reveal that he has undergone no pain, although the pressure on the side of the larynx has usually caused some discomfort. Slight soreness of the throat is not uncommon for one or two days after the examination, but this is seldom severe, and is always relieved by an aspirin gargle or a throat lozenge.

CONTRA-INDICATIONS

The few contra-indications to gastroscopy include those cases where the examination is not possible or justifiable on account of severe illness, especially if associated with dyspnœa. Such cases are beyond the scope of gastric investigation of any kind. Certain local conditions preclude the passage of the gastroscope, such as carcinoma or stricture of the œsophagus or cardiac orifice, gross curvature of the spine, aneurysm of the aorta, intrathoracic neoplasm. Cirrhosis of the liver or congestive heart failure with possible œsophageal varices also contra-indicate the instrumentation. After recent hæmatemesis gastroscopy is inadvisable on account of the risk of air embolism. The examination is also preferably not undertaken during an exacerbation of ulcer pain, as distension of the stomach may possibly cause a perforation of the softened base of an active ulcer. In expert hands, and with only slight inflation of the stomach, the latter two conditions need not absolutely preclude gastroscopy if the circumstances of the case make it a matter of necessity. The one great danger of the older method of gastroscopy—that of perforation of the œsophagus, with fatal mediastinitis—has been completely excluded by the advent of the flexible instrument.

Used with proper regard for contra-indicating circumstances, and with strict adherence to the dictum that no force must ever be used in the manipulation, gastroscopy with the flexible instrument is a method of investigation as completely without danger as the introduction of an ordinary stomach tube.

Henning has performed in his Klinik at Leipzig over 2000 gastroscopies without accident, and other clinicians both abroad and latterly in this country have adopted gastroscopy as a routine method of investigation. *Any* method of endoscopy, whether of the œsophagus, trachea, stomach, bladder, or rectum, is liable to accident in clumsy or unsympathetic hands. In the diagnosis of disease of the stomach gastroscopy is now to be regarded as corresponding in safety and in usefulness to these other older-established methods of instrumental investigation.

ENDOSCOPIC APPEARANCES OF THE STOMACH

The gastroscopic appearances described in these pages are illustrated by a number of paintings of actual cases made by an artist. The views were sketched in the operating theatre as the cases were demonstrated through the gastroscope.

The Normal Stomach.—The shape of the normal stomach has already been dealt with. In colour the mucosa has the same pink shade as that of the buccal mucous membrane, but is not so pale (*Fig.* 346). No blood-vessels can be seen, and the surface is glassy smooth, while the mucus of the ' Schleimsee ' forms a clear limpid pool. The rugæ are clear-cut straight folds, and the troughs between them are quite flat. The height of the folds varies with the degree of distension of the stomach. In the almost empty stomach folds are present over the whole of the surface, except that of

the lesser curve and pyloric antrum. The folds on the anterior wall are few and low, and are soon abolished by slight inflation of the viscus; those on the posterior wall are more marked, but can be abolished by full distension with air. The longitudinal folds marking the greater curve are the most pronounced of all, and can still be seen in the distended viscus.

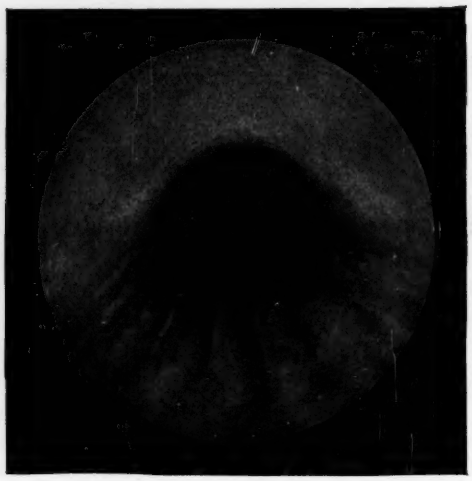

FIG. 346.—View of normal stomach looking towards the pylorus.

Gastritis: Hypertrophy: Atrophy.—Chronic inflammatory changes in the mucosa are indicated by general heightening of the colour to a deep red, and at the same time the surface loses its mirror-like smoothness and assumes a matt texture. The pool of mucus is no longer clear, and the secretion hangs on to and between the folds of mucosa in a thick sticky film. Areas of patchy congestion of the mucosa can be seen, and there may be hæmorrhagic extravasations into the submucosa according to the degree of inflammation. The rugæ in uncomplicated gastritis—such as is associated with the presence of a carcinoma—are dull and œdematous, but remain relatively straight.

These are the appearances of *gastritis* alone. It has become habitual to subdivide gastritis into a greater or lesser number of types, and to include, at the least, atrophic, hypertrophic, and catarrhal forms, while Moutier subdivides these into numerous subgroups according to associated manifestations such as hæmorrhages, erosions, etc. As the result of a still small experience of gastroscopy, the author is coming to the conclusion that there is only one form of chronic gastritis—the so-called 'catarrhal' type described above. The conditions named 'atrophic gastritis' and 'hypertrophic gastritis' appear to be combinations of atrophy or hypertrophy of the epithelium with a quite secondary inflammatory change of greater or less degree. Cases have been examined in which there was epithelial hypertrophy or atrophy without any of the signs of inflammation at all, others in which the inflammatory element has been slight, and others in which it has been considerable, while the degree of atrophy or hypertrophy in the various cases has borne no apparent relation to the degree of gastritis.

The appearances of *hypertrophy* of the mucosa are: A bright but uniform red colour of the surface, which is still shiny; close-up inspection shows that the surface is no longer flat but exhibits a very fine smooth velvety nodulation or mammillation, the 'Höckerung' described by Henning, indicative of the epithelial hyperplasia; the mucus in a case without inflammatory changes is clear, but the most obvious characteristic is the redundancy of the rugæ, which are steep and tortuous so that those on the anterior wall are still present when the viscus is moderately distended, while the posterior ones persist in spite of full inflation. These appearances and those of gastritis described above may be superimposed to form the so-called 'hypertrophic gastritis'. It is in this combination of circumstances that multiple small acute erosions commonly occur (Moutier's 'gastritis erosiva'). These are clear-cut, dull-yellow areas, almost level with the general surface of the mucosa. It seems that both hyperplasia (with hyperacidity) *and* gastritis are necessary before the erosions occur. Whereas in hyperplastic gastritis with or without a chronic ulcer small acute erosions are often seen, these are never seen after a course of treatment when the gastritis has subsided but the hyperplasia remains. In health the resistance of the epithelial cells to digestion by their own secretion appears to afford sufficient protection against even a raised acidity. If, however, the cells are devitalized by inflammatory changes, their defence is impaired and peptic erosions may occur as a result. In the absence of severe gastritis, gastric hyperplasia is invariably associated with a high test-meal acidity. Superimposed inflammation, however, appears to depress the secretory activity of the epithelial cells less than their resistance to auto-digestion, for erosions occur without much diminution of the hyperchlorhydria. Duodenal ulcer (demonstrated radiologically) is the type of erosion most commonly associated with 'hyperplastic gastritis'. The symptoms, however, commonly attributed to the ulcer appear to be mainly referable to the gastric condition, for cases with 'duodenal' symptoms of pain relatively late after meals, relieved by food, bismuth, or alkalis, all have the same characteristic endoscopic appearances, whether an ulcer is demonstrated by the barium meal or not.

The appearances of mucous *atrophy* of the stomach are well seen in pernicious anæmia. In this disease the mucosa is very pale and of a slightly yellowish tinge. The rugæ are ill-defined or absent, the interior of the stomach being a uniform flat surface, rather translucent in appearance, and the branching vessels of the submucosa can be seen through the attenuated superficial layers. The presence of inflammation may be found in the dull œdematous change in the surface and the presence of occasional hæmorrhages, but, again, the degree of atrophy and the degree of inflammation bear no apparent relation to one another.

These observations are entirely in agreement with Hurst's postulation of the congenital hypo- and hyper-sthenic types of stomach which are more prone than the normal viscus to attacks of gastritis, and which in the hypersthenic stomach may give rise to peptic ulceration.

Rare Conditions.—Interesting and unusual conditions may be revealed by gastroscopic examination to elucidate cases of otherwise symptomless hæmatemesis, in which all other investigations are negative. The examination of course is performed some time after the bleeding has ceased. One such case showed a mild degree of diffuse atrophic change with an area of mucosa which was swollen and thickened in bullous fashion, with curious folds unlike the normal gastric rugæ, in that they were short and irregularly arranged. Any possibility of a malignant

condition was excluded by the fact that the folds were soft and flexible and that peristalsis passed normally over this area of the stomach wall. The case was one of a localized area of *polyposis* in an atrophic mucosa. There had been two hæmatemeses separated by a ten-year interval, the second being severe enough to reduce the hæmoglobin to 30 per cent of normal.

Another case of unexplained hæmatemesis, that of an otherwise symptomless young man who had had six severe bleedings in two years, showed a condition of diffuse *gastrostaxis*. The whole of the mucosa was œdematous and dark brownish-red in colour, and was studded all over with submucous hæmorrhages. Blood examination in this patient revealed a very low platelet count which was considered to be the cause of the bleeding, although no hæmorrhages had occurred elsewhere.

Innocent tumours are occasionally seen endoscopically, though they rarely give rise to symptoms. Two examples have occurred in my series, and the endoscopic

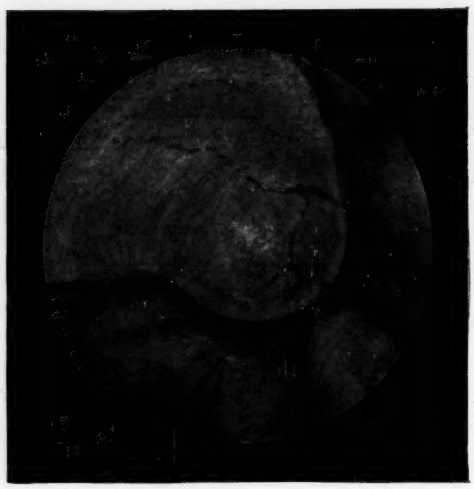

FIG. 347.—*Case* 18. Innocent tumour of stomach, probably a myoma.

picture of one of these is included. *Fig.* 347 shows what is probably a myoma. It was discovered accidentally during endoscopic examination of the stomach in a case of severe anæmia. There were no gastric symptoms. The tumour shows the characteristic smooth rounded even contour, and a dilated superficial vein can be seen through the stretched and thinned mucosa covering it. In another case a similar tumour was discovered in the pyloric region. The patient had had occasional attacks of abdominal pain and latterly a regular pain after food. The general characters of the tumour were similar to those of the previous case, but in addition there was an acute peptic erosion of the attenuated epithelium on the summit of the swelling. The importance of these cases lies in their differentiation from carcinoma. In the second case the filling defect was observed radiologically, and the suspicion of carcinoma had been raised and apparently confirmed by the test-meal report of achlorhydria. The true nature of the condition was only discovered on gastroscopy.

Ulcer.—Peptic ulcers occur in a stomach which is already the seat of diffuse hyperplastic and inflammatory changes in the mucosa. All stages of ulceration may

be found between the acute epithelial erosions described above and the typical chronic ulcer. The flat indurated base and clear-cut margins of the latter are well recognized and need no amplification here, but the gastroscope shows in the living subject the contrast between the bright-red mucosa round the ulcer and the yellow colour of the base (*Fig.* 348). Another feature of the gastroscopic view is the ring of œdematous mucosa round the crater of an active ulcer, an appearance which is lacking in autopsy specimens. The appearances of the ulcer vary according to its activity or stage of healing. *Figs.* 349–352 show the changes well. They are paintings of the gastroscopic appearances of the same ulcer under medical treatment. *Fig.* 349 shows the active ulcer, a deep circular crater with a wide area of smooth œdematous congested mucosa round it. The second picture (*Fig.* 350), taken one month later, shows the changes indicative of healing. The ulcer is now a shallow crater, due to the subsidence of the surrounding œdema, with

FIG. 348.—*Case* 51. Chronic peptic ulcer on lesser curve (healing). Note: clear-cut edges of yellow base; radiating cicatrices in surrounding mucosa; 'hitched-up' appearance of incisura angularis.

newly-formed epithelium encroaching from the edge, which is no longer circular and undermined but crenated and shelving down to the level of the ulcer base. It will be noted that the red granulating surface typical of a healing ulcer elsewhere does not occur with peptic ulceration. The most striking change, however, lies in the radiating folds of mucosa surrounding the ulcer. These are due to submucous cicatrization in the region of the crater which has taken the place of the inflammatory elements. In the third picture, three weeks later still (*Fig.* 351), the stellate area of mucosa is now seen to be projecting somewhat into the cavity of the stomach. This is due to the contraction of the scar tissue in the wall of the stomach under the base of the ulcer, as well as in the surrounding submucosa, so that the ulcer and surrounding mucosa are bunched up and invaginated into the lumen. This is an indication that the ulcer is not adherent in the lesser sac—a point of prognostic importance. The fourth picture, three months later (*Fig.* 352), shows the ulcer completely healed. The scar tissue is fully contracted and the mucosa has evened itself out again over the surface.

The apparent size of an ulcer seen endoscopically varies with the distance from the objective of the instrument. Thus one must not be misled by the apparent small size of pyloric ulcers compared with the ulcer high up on the lesser curve.

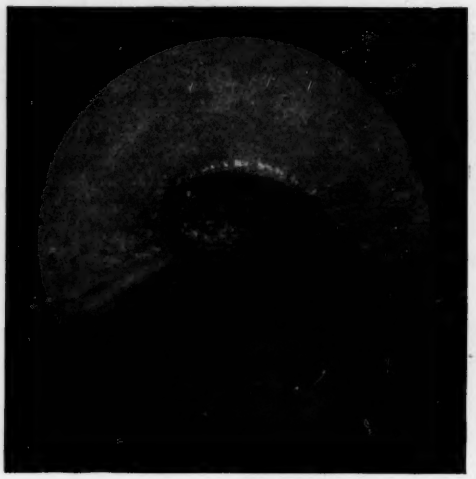

FIG. 349.—*Case* 12. Atrophic chronic peptic ulcer, untreated. Note: surrounding inflamed mucosa—œdematous, but with no radiating folds indicative of healing.

FIG. 350.—*Case* 12. Same ulcer after 4 weeks' medical treatment. Note signs of healing: radiating cicatrices; shallow crater; crenated border.

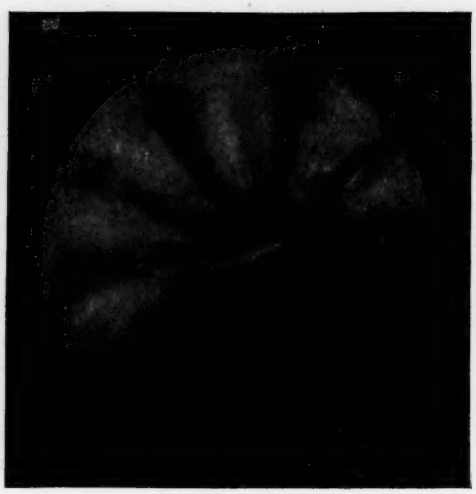

FIG. 351.—*Case* 12. Same ulcer after 7 weeks' treatment. Note: elevation of area of ulceration into the lumen (*see* text); crater almost disappeared.

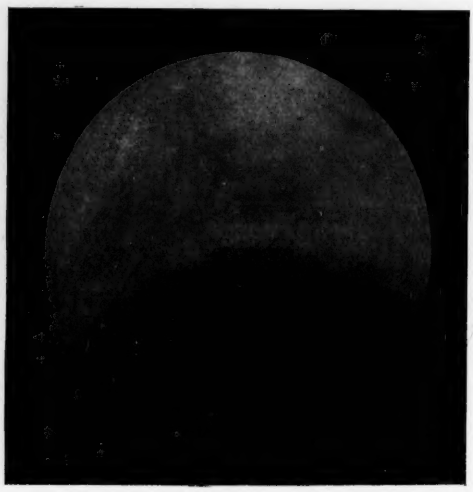

FIG. 352.—*Case* 12. Same ulcer healed. View taken three months after discharge from hospital. Note: mucosa of region of old ulcer has become level again—the stellate scar is almost abolished.

With some experience it is often possible to estimate the size of an ulcer by its relation to the size of the bow of the lesser curve or the diameter of the pyloric antrum. More important and easier is to report on the increase or decrease in size of any given ulcer on a second inspection after a course of therapy.

The degree and extent of cicatrization of the stomach wall near an ulcer can be observed gastroscopically, and the presence of adhesions in the lesser sac can be judged from the 'hitched up' appearance of the ulcer and its fixity in spite of

FIG. 353.—*Case* 37. Acute gastro-jejunal ulcer. Note: pylorus; valvulæ conniventes of jejunum; acute peptic erosion almost level with gastric mucosa.

FIG. 354.—*Case* 60. Chronic gastrojejunal ulcer. Note: acute extension down the jejunum; excrescence of grossly inflamed gastric mucosa overhanging ulcer crater; efferent loop of jejunum; pyloric antrum (on left).

FIG. 355.—*Case* 4. Part of the edge of a gastro-jejunal stoma with underlying chronic anastomotic ulcer. Note: efferent loop of jejunum with valvulæ conniventes; polypoid excrescence of grossly inflamed gastric mucosa (the ulcer lay beyond this).

manipulation of the abdomen or tilting the patient. At a second inspection, if, although the ulcer is healing, it is not level with the gastric wall or even invaginated a little as described above, then its adherence in the lesser sac is confirmed. These points are of value in giving a prognosis of the prospects of cure by medical means.

Ulcers on the anterior or posterior walls tend to be hidden by the redundant folds of hypertrophied mucous membrane, and are less obvious than the lesser-curve ulcers, which make a striking contrast to the normal smooth contour of this region. In a J- or U-shaped stomach an ulcer of the lesser curve of the pyloric antrum cannot be directly seen, but in such cases there is a peculiar and characteristic notching of the normally rounded contour of the incisura angularis. This appearance has often led the author to suspect the presence of such an ulcer, which after some manipulation and tilting of the patient has at length been brought into view.

Anastomotic Ulcer.—Endoscopic examination of a stomach which has been operated upon, usually for the performance of gastro-enterostomy, presents special difficulties on account of the distortion of the stomach which may be present, but chiefly because the viscus can seldom be satisfactorily distended with air, which tends to escape through the stoma. Moreover, the presence of a double loop of gut between the stomach and the already prominent vertebral column tends still further to lift the posterior gastric wall forward, thus turning the stoma away from the direction of the instrument, which lies in the paravertebral depression in front of the left kidney. It was on this account that the balloon was designed, and this has been found to be of great help in obtaining a view of the stoma. The opening itself is instantly recognizable (*Figs.* 353–355), for the transverse valvulæ conniventes of the jejunum seen through the stoma are unmistakable. The instrument can often be made to pass for some inches along the efferent loop of intestine, and with the aid of the balloon the jejunal mucosa can be inspected. In spite of the difficulties it is possible to obtain a satisfactory view of the stoma in the great majority of cases. It must be admitted, however, that in the author's experience stomata without ulceration are much easier to visualize than those with an ulcer. It is therefore much easier to establish the absence of a gastro-jejunal ulcer when suspected than directly to observe its presence. The reasons for this lie in the extra distortion of the stomach and the stoma by the ulcer, and also in the intense gastritis that occurs near the ulcer, which becomes hidden from view by the overhanging œdematous folds of hypertrophied mucosa. This latter handicap can nevertheless be turned to good account in diagnosis, for a localized patch of intense gastritis on one side of the stoma, in contrast with a lesser degree of gastritis elsewhere, is strong evidence of the presence of an ulcer, the crater of which may be hidden from the objective of the instrument (*Figs.* 354, 355).

'Post-operative' Gastritis.—Although the great majority of cases *with symptoms* after gastro-enterostomy have a greater or less degree of gastritis, I cannot subscribe to the view of some authors that gastritis is the invariable result of the performance of gastro-enterostomy. In spite of the fact that all my cases have had symptoms which have been the indication for the gastroscopy, I have seen three uninflamed stomata. In each the gastro-jejunal junction was a thin line where the two types of mucosa were in apposition. The gastric and jejunal surfaces were of uniform colour and possessed the normal highly reflecting surface, although the mucosa of the stomach showed the tortuous redundant rugæ indicative of hyperplasia.

In the interpretation of post-operative gastroscopic appearances, it is well to remember that the operation was performed for peptic ulceration complicating chronic gastritis in a stomach with a hyperplastic mucosa. Even under medical

treatment of such a condition the hyperplasia persists though the inflammation may subside, and it is therefore not surprising that after gastro-enterostomy the stomach still exhibits a hyperplastic mucosa which may again become the seat of

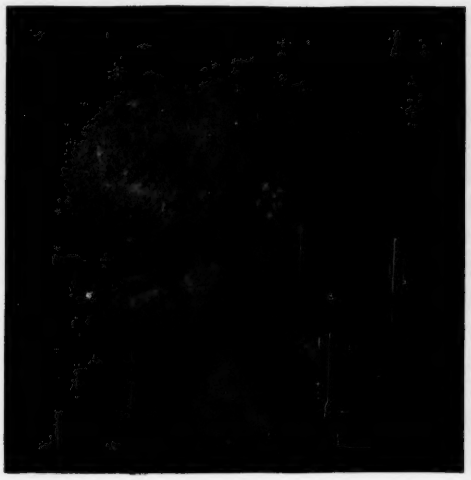

FIG. 356.—*Case* 53. Early carcinomatous ulcer on anterior wall of stomach. Note: infiltration of surrounding submucosa, which is raised into low mounds by the growth beneath.

FIG. 357.—*Case* 7. Ulcerating 'massive' carcinoma of greater curve. Note: raised everted edge of growth; sloughing base of ulcer.

FIG. 358.—*Case* 24. Upper limit of carcinoma of lesser curve. Note: 'rigid' appearance; submucous hæmorrhages; nodules of growth in submucosa.

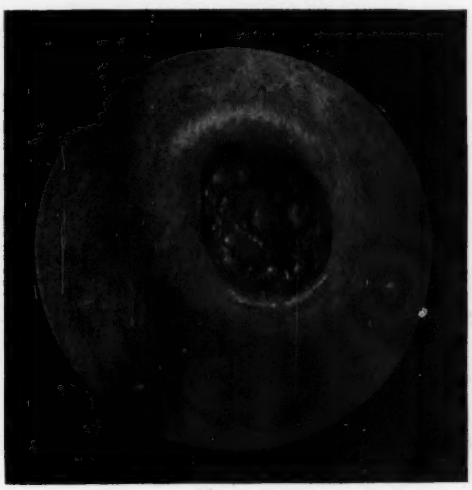

FIG. 359.—*Case* 23. Carcinomatous ulcer of lesser curve. Note: surrounding infiltration of mucosa; nodular sloughing base.

a recurrent or chronic inflammatory change. It is indeed not claimed for gastro-enterostomy that it cures or prevents the recurrence of the gastritis which is the underlying cause of the peptic ulcer, but that it reduces the gastric acidity to a

point at which the devitalized epithelial cells can resist its eroding action. The operation is only to be considered as removing the dangerous and troublesome complication of ulceration from the otherwise relatively harmless disorder of hyperplastic gastritis. But to ascribe the antecedent gastritis to the operation is hardly in accordance with the pathological sequence of events.

Carcinoma.—The superficial appearance of carcinomata of the stomach also needs no description here, except in regard to the special additional points which can be observed gastroscopically, by virtue of the fact that the growth is alive and the blood circulation is going on within it (*Figs.* 356–359). Thus the massive carcinoma, with or without central necrosis and ulceration, is of a high colour, and hæmorrhages may be seen within it. Occasionally pale submucous nodules of growth can be seen in contrast with the red gastric mucosa near the tumour, an appearance not observed in colourless autopsy specimens. One can observe the rigid character given to the stomach wall by the malignant infiltration in contrast to the soft flexible surface elsewhere, which alters in shape with the degree of distension and with respiratory and cardiac movements.

The gastroscope is unfortunately of little use in assessing the operability of a growth. Not only are metastases in the liver or lymph-nodes beyond its field, but the area of stomach wall above a visible growth cannot be pronounced free from infiltration. Only the mucosal aspect is visible, and the spread of carcinoma in the subperitoneal lymph-plexus of the stomach cannot be detected from within.

Gastroscopy is contra-indicated with a growth at the cardia. For this reason alone X-ray examination is a necessary preliminary to gastroscopy if this condition is suspected.

THE CLINICAL VALUE OF GASTROSCOPY

It is obvious that direct inspection of the living gastric mucous membrane is scientifically a matter of the first importance for that development of our knowledge of the pathology of the stomach which alone can lead to improved methods of clinical therapy. The object of this paper, however, is to show that gastroscopy has a useful place in the investigation of ordinary clinical cases notwithstanding the invaluable help of modern radiological technique. Further, that in many cases gastroscopic examination can only be omitted at the expense of the patient, either by submitting him to unnecessary laparotomy or to inadequate treatment through lack of a sufficiently accurate diagnosis. It must be emphasized, however, that gastroscopy is the last investigation that should be made upon a patient, and that, for reasons which will subsequently be discussed, it should not be performed without previous radiography of the stomach.

Carcinoma.—If in the investigation of a case, after consideration of the clinical evidence, the radiological report, and the results of test-meal and occult-blood examinations, a diagnosis is definitely established upon which operation is indicated, such as carcinoma with a gross filling defect, then gastroscopy is not required. In a number of cases, however, there is an element of doubt in the diagnosis largely due to the circumstantial nature of the evidence, except that of the radiograph. The interpretation of the test-meal and occult-blood examinations is notoriously problematical, and in the doubtful case the onus of diagnosis is therefore thrown upon the radiologist. In these circumstances the important

decision whether to operate or not often turns on the radiologist's opinion of a suspicious area of the stomach, about which he cannot be certain. In the past it has been a not uncommon occurrence for a patient, suffering from no more than an atrophic gastritis, but under suspicion of carcinoma, to be submitted to laparotomy, on the plea that it is better to operate and to be mistaken than to miss a growth while it is still operable. Gastroscopy now provides the means whereby such mistakes can be averted, and is most strongly indicated in cases of doubtful carcinoma where the radiologist cannot give absolute confirmation of the diagnosis. If the doubtful area of the stomach suspected by the radiologist can be brought before the objective of the gastroscope, and the gastroscopist can say whether this has been done or not, then a definite answer can be given to the question—is there or is there not a carcinoma present? In this way a diagnosis of carcinoma can be established in the early, still doubtful case. It is generally accepted that the chief factor in any improvement that may be achieved in the treatment of malignant disease, especially of the stomach, is early diagnosis, and it is in this field that gastroscopy is so valuable an addition to the available methods of investigation.

In the case with symptoms referable to the stomach and a completely negative radiograph, gastroscopy is nevertheless still indicated. Apart from the desirability of establishing the degree and type of gastritis in such circumstances, cases do occur, although rarely, in which an early carcinoma or a posterior-wall ulcer has been overlooked on radiological examination. The records of any hospital will show cases which, having been admitted for gastric symptoms and discharged as 'gastritis' after negative results from X rays and test-meal, have been re-admitted some time later with advanced carcinoma of the stomach. Such a case came to the post-mortem room recently in which a carcinoma of the pylorus had been undiagnosed at a previous investigation because the presence of a gastro-enterostomy opening made years previously had diverted the barium meal and prevented the radiologist from outlining the growth. Gastroscopic examination at the time of investigation would probably have revealed an early carcinoma which might have been successfully removed.

Gastritis.—If, happily, a carcinoma be not found on gastroscopy and the radiological report be confirmed in this respect, great importance is still to be attached to the identification of the type of generalized disease of the mucosa which is giving rise to the symptoms. The view that peptic ulceration is a sequel of an untreated 'acid' gastritis in a hyperplastic stomach is rapidly commanding acceptance. An accurate diagnosis of this condition before the stage of ulceration, only possible by means of the gastroscope, will enable prophylactic measures to be taken against the supervention of ulceration. Alternatively, in the hypoplastic type of stomach, gastritis is now established as a primary etiological factor in the production of certain forms of anæmia, so that attention to the stomach is of the first importance in the treatment of, or prophylaxis against, this type of disease. Also, the view is gaining ground that carcinoma of the stomach, as with carcinoma elsewhere, may be a possible consequence of the chronic irritation of gastritis in an atrophic mucosa, although this is as yet non-proven. The gastroscope has done much towards the restoration of 'gastritis' as a definite pathological entity. It now appears that this much neglected clinical condition may be fraught with serious consequences, so that in its diagnosis and treatment gastroscopy may prove to be a major influence in the reduction of the incidence of its better recognized and more dangerous sequelæ.

Ulcer.—As with carcinoma, if a gastric ulcer is found on X-ray examination of a case in circumstances which indicate operative treatment, again gastroscopy is not required. But if doubt as to the advisability of operative treatment exists, having regard to the poor general condition of the patient or to the possibility of cure by medical means, then gastroscopy can obtain the evidence whereby the exact nature of the ulcer can be established and the correct course of treatment determined. The gastroscopic appearances of the acute erosion, the chronic ulcer, or the healing ulcer are for the most part well-defined and easily recognized, and a confident opinion on the case can usually be given by the gastroscopist.

The establishment of an exact diagnosis is not, however, the limit of usefulness of the gastroscope in the field of peptic ulceration. If an ulcer is to be treated medically, then the gastroscopist can inform the physician of the effect of his treatment both on the ulcer and on the associated gastritis. It is often a very difficult matter to know when a gastric ulcer is healed and the patient can be allowed to return to normal life. X rays do not really demonstrate an ulcer—they outline a crater formed, not by the ulcer, but by the œdematous mucosa on either side of it. This œdema subsides rapidly under treatment, and the barium meal may fail to show a crater long before healing is complete, particularly if the ulcer lies to one or other side of the lesser curve. An instructive case of lesser-curve ulcer occurred recently, which was considered healed after five months' medical treatment. Repeated radiographs had shown the crater to have disappeared, yet the ulcer perforated a fortnight after the patient had been discharged from hospital! Gastroscopy, by rendering the ulcer open to direct inspection, now enables the stages of healing to be observed and the completion of the process to be confidently determined (*see Figs.* 349–352). If, on the other hand, the ulcer is not responding to treatment, then gastroscopy will establish this fact beyond question, and surgical measures can be undertaken without loss of time in useless therapy.

The supervention of a malignant change in a chronic ulcer is not always visible to the naked eye, and therefore cannot always be diagnosed at a first gastroscopic examination. Where, however, considerations of grave operative risk justify a delay in radical treatment of a doubtful case, a second inspection of the ulcer after a month's medical treatment will reveal whether it is healing and therefore innocent, or progressing and therefore probably malignant. The value of this method is exemplified in the type of necrotic ulcer occurring in the atrophic senile stomach, which often closely resembles a carcinoma in the shortness of the history, the loss of appetite, loss of weight, the associated achlorhydria, and the large size of the ulcer. These cases, however, readily respond to medical therapy, so that the decision as to malignancy or innocency need not be delayed for longer than a month. In these old patients such a delay in operative treatment of the malignant case is more than compensated by the satisfactory healing of the ulcer in other cases, where an extensive operation might have led to a fatal issue.

The value of gastroscopy in the assessment of new methods of therapy such as the recently introduced histidine treatment is evident, but it is beyond the scope of this paper to enlarge on this aspect of the method.

Gastro-jejunal Ulcer.—In the diagnosis of gastro-jejunal ulceration gastroscopy is an investigation of special value. The presence of the stoma greatly diminishes the value of the test-meal, and it is well known that anastomotic ulcers are

notoriously difficult to show radiographically by the barium meal. The reports of *Cases* 4 and 60 are a striking illustration of this. In each of these, X-ray examination had produced negative results on more than one previous occasion; and even immediately preceding the observation of the ulcer through the gastroscope, the screen had failed to find evidence of any pathological change in the stoma. Moreover, in this type of case the clinical picture of gastro-jejunitis without ulceration is so similar to that of an actual anastomotic ulcer that clinical diagnosis is a matter of extreme difficulty. In such a case, if the stoma can be brought into the field of the gastroscope a definite observation can be made as to the presence or absence of a gastro-jejunal ulcer. It has been pointed out that the gastro-enterostomized stomach presents special difficulties to the gastroscopist, but in spite of this a definite diagnosis can be made in the great majority of cases.

RELATION OF GASTROSCOPY TO RADIOLOGY OF THE STOMACH

The advent of gastroscopy does not in any way detract from the value of radiology in gastric investigation. The barium meal often suffices alone to establish the diagnosis, and must always be the routine method of examination of the stomach. Moreover, gastroscopy, to be used safely and to the best advantage, will always be to a certain extent dependent on previous radiology, the two methods of investigabeing complementary to each other in their results. X rays demonstrate the size, shape, position, and movements of the stomach, and reveal gross filling defects or deep ulcers. The gastroscope can extend this description to include the more minute characters of the mucosa—the presence of inflammation, hyperplasia, atrophy, hæmorrhages, or erosions; or the radiological silhouette of a suspicious area can be amplified by a description of the colour, texture, and surface configuration of the lesion. In this way radiology and gastroscopy combine together to provide a complete and accurate description of the stomach, so that, in the words of Henning, an exact morphological diagnosis can now be established in every case of gastric disease.

STATISTICAL ANALYSIS OF CASES

The first sixty cases gastroscoped at the London Hospital up to June, 1936, have been summarized and included as a table to illustrate the general use of gastroscopy (*see* pp. 494–500). The examination was not done as a routine, but only in cases where there was a definite indication for its employment. It will be seen that ten cases fall into the category in which there has been suspicion of carcinoma, either supported or not excluded radiologically, but in which no carcinoma has in fact been present. In most of these cases gastroscopy was a decisive factor in the avoidance of an exploratory operation. In three of them a suspicious ulcer was visible on the screen, but its innocent nature was established by the gastroscope either at once or after a short course of therapy. On the other hand four cases of dubious carcinoma were confirmed on direct inspection.

Three patients with symptoms of ulcer and supporting radiological evidence such as 'spasm' were found to be cases of gastritis only.

Two of these 'negative' cases had actually, in the past, had a fruitless laparotomy, so suggestive were the symptoms, while in two more operation was performed

in spite of contra-indicative gastroscopic findings, in each case with negative results (*Cases* 6 and 51).

Sixteen cases of doubtful gastro-jejunal ulcer were examined; the stoma was visualized in fourteen of these, and in eight cases anastomotic ulcer was definitely excluded. In six cases (*Cases* 4, 8, 10, 22, 37, and 60) an active ulcer was found, on three occasions by direct inspection, and in the other three cases by deduction from the presence of a localized polypoid excrescence of highly inflamed gastric mucosa at one part of the stoma. Of the three ulcers directly observed, two were fairly large acute erosions of the mucosa on the gastric side of the stoma, and one was a deep chronic ulcer. The three ulcers inferred from the polypoid excrescence of gastric mucosa over them were *ipso facto* chronic ulcers. In all, therefore, there were four cases of chronic gastro-jejunal ulcer, of which only one was directly visible. The cause of the symptoms in the sixteen unsatisfactory cases after gastro-enterostomy therefore was: Eight cases—persistent severe hyperplastic gastritis. Two cases—hyperplastic gastritis with acute gastro-jejunal erosion. Four cases—chronic anastomotic ulcer, also with gastritis. Two cases—normal gastric mucosa; normal stoma; symptoms not due to stomach.

A lesion in an unusual situation (other than gastro-jejunal ulcer) has been discovered in four cases (*Cases* 9, 21, 53, and 59) where the condition was not apparent on X-ray examination. Three of these were peptic ulcers on the posterior wall and one was an early carcinomatous ulcer on the anterior wall (*Case* 53). In two cases the presence of an ulcer which was doubtful radiologically has been confirmed.

SELECTED CASE REPORTS

Although the tabulated list of cases on pp. 494–500 gives some indication of the clinical value of the gastroscope, perhaps the following brief notes of some of the more interesting cases in the series provide a better demonstration of its use.

Case 4.—J. L. G., male, aged 53. Gastro-enterostomy in 1923 for duodenal ulcer. Symptoms have persisted since operation. Re-admitted in 1934 for investigation—? gastro-jejunal ulcer, with negative results from X rays and test-meal. Symptoms progressed. Admitted again in 1935 for further investigation, in poor condition. Again test-meal showed absent HCl and X rays showed a "patent freely-working stoma" with no evidence of ulceration. Gastroscopy showed a localized polypoid excrescence of highly inflamed mucosa projecting from the gastro-jejunal junction (*see Fig.* 355). The deduction was made that a gastro-jejunal ulcer was present on the jejunal side of the stoma beyond this area. At operation by Sir James Walton a large adherent gastro-jejunal ulcer was found and partial gastrectomy was performed. Examination of the specimen showed the ulcer rather on the jejunal side of the stoma under cover of the œdematous mucosal projection that had been observed gastroscopically.

Case 6.—E. S., female, aged 46. Long history of indigestion, clinically due to visceroptosis. Test-meal showed 0·18 per cent free HCl. X rays showed evidence of duodenal ulcer. Gastroscopy showed a moderate degree of gastritis in a rather atrophic mucosa. The opinion was given that duodenal ulcer was very unlikely in the absence of hyperplastic gastritis. Operation, however, was done as the X-ray findings were definite. No ulcer was discovered.

Case 10.—L. D., male, aged 48. Gastro-enterostomy eight years previously. Had been symptomless since operation until admitted with acute onset of diarrhœa, foul flatulence per mouth, vomiting of fæculent material, and rapid wasting. At operation a gastro-jejuno-colic fistula at the site of ulcer was found, but the condition of the patient prohibited anything further than the separation of the colon from the anastomosis. Patient did well after operation and had no symptoms as before, but gastroscopy eleven months later showed evidence of

another 'silent' gastro-jejunal ulcer. Strict medical treatment was commenced. Ten months later the scar of a healed gastro-jejunal ulcer was observed. Treatment relaxed.

Case 20.—J. W., male, aged 53. Twenty-eight years' history of indigestion, latterly increasing in severity. Test-meal showed 0·31 per cent free HCl, but the radiologist found a " very unusual appearance " of the stomach on which he was unwilling to give an opinion. The case was considered to be one of visceroptosis, but gastroscopy revealed the pylorus apparently situated in the middle of the lesser curve, adjacent to an ulcer. At operation this was found to be indeed the case. An old-standing large lesser-curve ulcer in a U-shaped stomach had resulted in the adhesion together of the limbs of the U, with subsequent erosion and disappearance of the resulting septum between the two halves of the stomach. The pylorus was therefore left opening into the stomach about three inches from the cardia. Partial gastrectomy was performed. Did well.

Case 21.—A. C., male, aged 63. Gastro-enterostomy 6 months ago for high adherent lesser-curve ulcer. Symptoms of ulcer persisted, but X rays showed no evidence of it. Gastroscopy showed the ulcer high up near the cardia, smaller, but still unhealed. Patient was put on a stricter course of treatment, and a second gastroscopy 5 weeks later showed the ulcer to be healed. Treatment was relaxed and the patient has kept well since.

Case 26.—F. G., male, aged 51. Short history of indigestion. Test-meal showed raised acidity. X rays showed small lesser-curve ulcer. Patient admitted as suitable for trial of histidine therapy. Fourteen days later, before treatment began, patient was gastroscoped, but ulcer was found to have already almost healed in the interval.

Case 32.—R. L., male, aged 52. Fifteen years' vague history suggestive of visceroptosis. X rays showed a dubious area on the lesser curve, but this was discounted by the clinician, who considered the symptoms to be due to the visceroptosis. Gastroscopy showed an adherent lesser-curve ulcer. After one month's treatment a second inspection showed no evidence of healing. Gastrectomy was therefore performed.

Case 37.—H. H., male, aged 43. Had been well since gastro-enterostomy 9 years previously for duodenal ulcer until a recent attack of epigastric pain and vomiting. X rays showed a normal stoma but an abnormal pylorus suggestive of carcinoma. Gastroscopy showed this appearance to be due to extra-gastric adhesions, but a large acute peptic erosion was observed at the stoma (*see Fig.* 353). The patient was put on strict medical treatment and has remained well since.

Case 51.—W. J., male, aged 44. Short history of indigestion, vomiting, and some loss of weight. Test-meal showed diminished secretion and X rays showed an ulcer on the lesser curve. The physician and surgeon considered the case to be a malignant ulcer. Gastroscopy showed the ulcer with all the signs of healing (*see Fig.* 348). The other aspects of the case were felt to be so suggestive that operation was performed two weeks later. No evidence of carcinoma was found. Wedge resection and gastro-enterostomy was performed. The excised ulcer was found to be half covered with newly formed epithelium.

Case 52.—M. C., male, aged 64. Ten years' history suggestive of ulcer, with a recent change in symptoms to include vomiting, loss of weight, and severer pain. Test-meal showed complete achlorhydria and X rays showed an "uneven lesser curve—? carcinoma". Gastroscopy showed a lesser-curve ulcer with signs of healing, although a clinical diagnosis of carcinoma was considered probable in spite of the endoscopic appearance. In view of *Case* 51 and the fact that the patient was a very bad risk for operation, a course of medical treatment was given. A second gastroscopy 6 weeks later showed active healing of the ulcer. Carcinoma was therefore definitely excluded and operation avoided. Three months later a third gastroscopy showed the ulcer reduced to a tiny speck—just on the point of complete healing.

Case 53.—J. B., aged 48. Two years' vague indigestion. No abnormal physical signs: X-ray and test-meal normal. Gastroscopy revealed an atypical ulcer on the anterior wall adjacent to a rounded elevation of mucosa (*see Fig.* 356). The suspicion of malignancy raised was discounted for lack of supporting evidence in test-meal or X-ray or clinically. Patient discharged himself after three weeks' medical treatment and did not

return for five months—again with vague epigastric discomfort. Gastroscopy now showed a large sloughing ulcer in a mass of growth. Operation by Mr. Alan Perry revealed an inoperable carcinoma of the greater curve and fundus, adherent to the pancreas.

The interest of this unsatisfactory case is that the malignant character of the ulcer was really observed very early, but through lack of experience was not recognized. One is led to hope and expect that such an error would not be repeated, and that gastroscopy will in future be able to diagnose a carcinoma before any other method of investigation while the symptoms are slight.

Case 57.—C. P., male, aged 52. Gastro-enterostomy three years previously. Operated upon three months ago for perforation of a gastro-jejunal ulcer on the efferent loop of the jejunum, which was sutured. On gastroscopy the whole of the efferent loop of gut was clearly seen, but there was no trace of ulcer. The ulcer therefore had been an acute one and had completely healed after the suturing.

Case 60.—S. S., male, aged 56. Gastro-enterostomy for duodenal ulcer in 1933. Indigestion persisted after operation. Investigation by X rays and test-meal in 1934 showed no evidence of anastomotic ulcer. Re-investigation in 1935 produced the same negative result. In 1936 patient admitted for gastroscopy. Test-meal showed 0·15 per cent free HCl. X rays once more showed no evidence of a gastro-jejunal lesion. Gastroscopy revealed a deep chronic ulcer crater on the gastro-jejunal junction opposite the efferent loop of intestine (*see Fig.* 354). There was in addition an acute extension of the ulcer down the jejunum, while the gastric mucosa overhung the crater in the characteristic polypoid excrescence. The findings were confirmed at operation by Sir James Walton, and partial gastrectomy was successfully performed.

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1936

Case No.	Lesion	Test-meal (1 hr.) HCl per cent	X-ray Report	Notes on Case: Endoscopic Appearances
1	Distortion by band	0	'Spasm' of antrum	History very suggestive of ulcer, including hæmatemesis and melæna. X-ray supported the possibility. Operation avoided through gastroscopy *N.B.*—A previous laparotomy had been performed years ago for the same symptoms and no lesion found
2	Hyperplastic mucosa	0·26	Ptosis; no ulcer	History suggestive of ulcer. This excluded. X-ray confirmed. *N.B.*—Presence of hyperplasia without gastritis
3	'Catarrhal' gastritis	0	No abnormality	History suggestive of carcinoma. Test-meal suggestive. Recent hæmatemesis. Carcinoma excluded by gastroscopy. X-ray confirmed
4	Gastro-jejunal ulcer	0	Normal stoma; no ulcer seen	Gastro-jejunal ulcer diagnosed by gastroscopy (*Fig.* 355) (*case report in text*) in spite of negative test-meal and X-ray. Partial gastrectomy
5	Normal stomach	0	—	Case of unexplained vomiting; ? mesenteric adenitis. Gastroscopy excluded gastritis

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1936—*contd.*

CASE No.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE: ENDOSCOPIC APPEARANCES
6	Atrophic gastritis	0·18	Duodenal ulcer; ptosis	Gastroscopic opinion given that duodenal ulcer was very unlikely with atrophic mucosa. Operation performed. No ulcer found
7	Carcinoma	—	Carcinoma (some doubt)	Carcinoma observed. X-ray confirmed. Partial gastrectomy. (*See Fig.* 357)
8	Acute gastro-jejunal ulcer; gastritis	—	—	Clinical diagnosis of chronic gastro-jejunal ulcer excluded. Acute anastomotic ulcer seen. Patient put on strict medical treatment. Now much improved
9	Posterior-wall ulcer	—	No abnormality	Ulcer discovered on posterior wall on gastroscopy. Ulcer had failed to show radiologically on account of its position. Medical treatment instituted. Second gastroscopy: showed ulcer still present. Feeble patient. Treatment continued
10	Gastro-jejunal ulcer	—	—	Patient had had a gastro-jejuno-colic fistula as first symptom of a 'silent' gastro-jejunal ulcer. Colon separated off at operation—ulcer not then active. Gastroscopy suggestive of recurrence of gastro-jejunal ulcer — again without symptoms. Medical treatment commenced. Second gastroscopy 10 months later showed the scar of a healed anastomotic ulcer. (*Case report in text*)
11	Hyperplastic gastritis	0·18	No abnormality	History suggestive of ulcer. Negative X-ray confirmed. Treatment given for gastritis, with improvement
12	Lesser-curve ulcer	0·20	Showed ulcer crater	Gastroscopic control of medical treatment. Stages of healing observed (*see text*). Completion of healing determined. Treatment relaxed. (*See Figs.* 349–352)
13	Carcinoma	0	? Carcinoma (some doubt)	Gastroscope held up just within cardia by growth. Nil seen. Instrument used only as a sound. Operation: inoperable carcinoma
14	Hypertrophic gastritis	0·20	Spasm of pylorus; ? pyloric ulcer	Pyloric ulcer excluded (duodenal ulcer still possible). Treatment given for gastritis [*Continued on next page*

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1936—*contd.*

CASE NO.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE: ENDOSCOPIC APPEARANCES
15	Duodenal ulcer	0·15	? Juxta - pyloric ulcer	Pyloric ulcer excluded. Much spasm of antrum observed. Duodenal ulcer suggested. Course of histidine therapy. Second gastroscopy showed no improvement. Operation performed, adherent duodenal ulcer found. Gastro-enterostomy
16	Non - pathological gastro - enterostomy stoma	—	No abnormality	Hyperplastic mucosa. No gastritis. Whole stoma observed: ulcer excluded. Gastro-jejunal junction a thin, clearly marked line
17	Pernicious anæmia	0	No abnormality	Carcinoma suspected by blood in test-meal. This excluded by gastroscopy. U-shaped stomach. Mucosal atrophy noted.
18	Benign tumour; atrophic gastritis; anæmia	0	No abnormality	Carcinoma suspected. No response to treatment for anæmia. Malignant growth excluded by gastroscopy (*see Fig.* 347)
19	Carcinoma	0	Carcinoma	Gastroscopy to determine operability. Growth reported operable, as normal mucosa above it was sufficient for gastrectomy. At operation, subperitoneal lymphatic infiltration of whole stomach—inoperable *N.B.*—Gastroscopy useless to determine operability
20	Lesser-curve ulcer; gross distortion	0·31	Appearances inexplicable; ? carcinoma	Long vague history. Case considered clinically to be visceroptosis. Gastroscopy showed the ulcer and distortion. Partial gastrectomy
21	High posterior-wall ulcer; stitch ulcer near gastro-enterostomy	0	No ulcer seen; normal stoma	Ulcer observed. Also bunch of granulations near stoma — ? round a stitch. Medical treatment. Second gastroscopy showed ulcer healed. Patient discharged
22	? Gastro-jejunal ulcer	0·13	Normal stoma	Indirect evidence of gastro-jejunal ulcer—a localized area of gastritis at one part of the stoma. Medical treatment
23	Carcinomatous ulcer	0	Ulcer; ? carcinoma (some doubt)	Patient in poor health. Carcinomatous nature of ulcer confirmed. Medical treatment otherwise intended. Partial gastrectomy. (*See Fig.* 359)

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1936—*contd.*

CASE No.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE: ENDOSCOPIC APPEARANCES
24	Carcinoma	0	Carcinoma	Gastroscopy showed growth operable as regards mucosa above it. Laparotomy—extensive involvement of pancreas. Inoperable. (*See Fig.* 358)
25	Lesser-curve ulcer	0·11	Ulcer seen; ? malignant	Short history suggestive of carcinoma. Ulcer seen to be a typical peptic ulcer already healing. Operation nevertheless performed on other evidence. Non-malignant ulcer found. Cautery excision and gastro-enterostomy
26	Lesser-curve ulcer	0·12	Showed ulcer	Gastroscopy performed to observe effect of histidine therapy. Ulcer found to be almost healed before treatment commenced!
27	Distortion of stomach; ? cause	0	? Lesser-curve ulcer	First X-ray examination suggested ulcer. Repeat X-ray did not confirm this. Gastroscope held up on posterior wall by obstruction 2 in. within cardia. Gastritis. Obstructing agent not visible. No operation.
28	Gastro-jejunitis	—	No abnormality	Case suggestive of gastro-jejunal ulcer. Stoma well seen; ulcer excluded. Medical treatment
29	Hyperplastic gastritis; jejunitis	0	Normal stoma	Case suggestive of gastro-jejunal ulcer. Stoma well seen; ulcer excluded. Medical treatment. *N.B.*—Gastritis much more intense than jejunitis
30	Hyperplastic gastritis; distorted stoma	—	—	Case suggestive of gastro-jejunal ulcer. Abnormal funnel-shaped stoma seen as though pulled upon. Ulcer excluded
31	Distortion of stomach; normal stoma	0	? Pyloric lesion; inefficient stoma	Dyspepsia after gastro-enterostomy. Stoma well seen. No inflammation. Pyloric lesion excluded. Ridge observed due to band across lesser curve
32	Lesser-curve ulcer	0·15	Dubious; ? ulcer present	Ulcer demonstrated. Medical treatment. Second gastroscopy showed no improvement. Ulcer seen to be adherent. Partial gastrectomy
33	Gastro-jejunitis	0·1	No abnormality	Whole stoma seen. Mild inflammatory changes. Gastro-jejunal ulcer excluded

[*Continued on next page*

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1939—*contd.*

CASE NO.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE: ENDOSCOPIC APPEARANCES
34	Polyposis	0·1	No abnormality	A case of unexplained recurrent hæmatemesis (twice in ten years). Polyposis observed
35	Normal stomach; carcinoma of liver	0·09	Filling defects; ? carcinoma of stomach	Filling defects observed to be the result of pressure from without (liver). Operation—carcinoma of liver
36	Carcinoma	0	Dubious; ? carcinoma high up	Upper end of growth observed. Inoperable
37	Gastro-jejunal ulcer	0·1	Normal stoma; ? carcinoma of pylorus	Acute erosion seen on gastro-jejunal junction. Pylorus observed: carcinoma excluded. Medical treatment. (*See Fig.* 353)
38	Gastro-jejunitis	0	Tenderness over stoma; no ulcer seen	Case suggestive of gastro-jejunal ulcer. Whole stoma seen. Ulcer excluded. Medical treatment
39	Benign tumour; acute ulcer	0	Filling defect	Feeble patient. Case suggestive of carcinoma. This excluded. Acute ulcer seen on top of benign tumour. Operation avoided. Medical treatment
40	Hyperplastic gastritis	0	Normal stoma; ? carcinoma of pylorus	Gastritis observed. Failed to see into stoma, which was turned away from instrument. Pylorus seen. Carcinoma excluded. Medical treatment
41	Gastro-jejunitis	0·04	—	Whole stoma seen. Ulcer excluded. Marked hyperplastic gastritis; Mild jejunitis
42	Atrophic gastritis	0	Dubious; ? carcinoma of pylorus	Whole stomach seen. Carcinoma excluded
43	Lesser-curve ulcer	0·07	Ulcer demonstrated	Failed to see ulcer owing to distortion of stomach obstructing instrument. Another healed ulcer observed high up
44	Normal stomach	0·1	No abnormality	Case of unexplained abdominal pain and hæmatemesis. Whole stomach seen. Gastric lesion excluded
45	Lesser-curve ulcer	0·19	Carcinoma of lesser curve	Typical peptic ulcer observed. Second gastroscopy after one month showed little healing; much scarring; ulcer adherent. Operation—peptic ulcer. Partial gastrectomy
46	Hypertrophic gastritis	0·15	Filling defect; ? carcinoma	Whole stomach seen. Carcinoma excluded. Gastritis treated medically

TABLE OF 60 SUMMARIZED CASES OF GASTROSCOPY PERFORMED UP TO JUNE, 1936—*contd.*

CASE NO.	LESION	TEST-MEAL (1 HR.) HCl per cent	X-RAY REPORT	NOTES ON CASE: ENDOSCOPIC APPEARANCES
47	Hypertrophic gastritis; healed ulcer	0·2	Doubtful ulcer near stoma	Case of dyspepsia after gastro-enterostomy. Failed to see stoma. Very mild gastritis observed. Ulcer therefore improbable. Recently healed erosion seen
48	Distortion by band	0·18	Distortion; ? duodenal and lesser-curve ulcers	Lesser-curve ulcer excluded. Distortion by extra-gastric adhesions. Medical treatment
49	Gastrostaxis	—	—	Case of recurrent hæmatemesis (6 attacks in 2 years). Diffuse submucous hæmorrhages seen. (Blood-platelet count, 60,000)
50	Lesser-curve ulcer	0·06	Dubious; ? ulcer	Lesser-curve ulcer seen to be healing. Medical treatment
51	Lesser-curve ulcer	0·06	Ulcer demonstrated	Clinical suspicion of malignancy. This excluded by gastroscope. Ulcer seen to be healing. Operation nevertheless performed two weeks later. Typical peptic ulcer found half healed. (*See Fig.* 348)
52	Lesser-curve ulcer	0	Ulcer; ? carcinoma of lesser curve	Case very suggestive of carcinoma. Feeble patient. Typical peptic ulcer observed. Medical treatment. Second gastroscopy showed healing in progress. Carcinoma definitely excluded
53	Early anterior-wall carcinoma	0·09	No abnormality	Patient with vague symptoms. Ulcer observed adjacent to a swelling of the mucosa on anterior wall. Doubtful: ? ulcer; ? carcinoma. Second gastroscopy after 3 weeks showed little change. Patient discharged himself. 6 months later—a little more pain. Third gastroscopy showed extensive carcinoma. *N.B.*—With more experience this carcinoma could have been diagnosed extremely early. (*See Fig.* 356)
54	Atrophic mucosa	0	Dubious carcinoma of pylorus; normal stoma	Case of anæmia not responding to treatment. Short history, carcinoma suspected. Whole stomach seen. Carcinoma excluded. Normal gastro-enterostomy stoma seen
55	Hyperplastic mucosa	0·22	No abnormality	Case very suggestive of ulcer. This excluded. *N.B.*—Hyperplasia without gastritis

[*Continued on next page*

Case No.	Lesion	Test-meal (1 Hr.) HCl per cent	X-ray Report	Notes on Case: Endoscopic Appearances
56	Anæmia	0	—	Gastroscopy failed: S-shaped œsophagus; instrument would not enter cardia. Ulcer seen in œsophageal wall above cardia
57	Hyperplastic gastritis; healed gastro-jejunal ulcer	—	—	Case of recently perforated acute gastro-jejunal ulcer. Sutured. Stoma clearly seen. No recurrence of ulceration observed
58	Hyperplastic gastritis; acute erosions	0·17	Ptosis; no ulcer	Marked hyperplastic gastritis and multiple erosions seen. Medical treatment. Second gastroscopy showed much less gastritis, no erosions, but still marked hyperplasia of mucosa
59	Mucosal hyperplasia; healed ulcer	0·22	No abnormality	Recently healed ulcer seen on posterior wall. Hyperplastic mucosa, but very slight gastritis
60	Chronic gastro-jejunal ulcer	0·15	No evidence of ulcer	Case of repeated negative investigations of persistent symptoms after gastro-enterostomy. Chronic ulcer observed on gastroscopy. Partial gastrectomy. (*See Fig.* 354)

I should like to express my appreciation to the Association of Surgeons of Great Britain and Ireland of the honour of being elected Moynihan Fellow for 1936 in order to undertake this work.

I wish to thank also the authorities at the London Hospital for permission and facilities to carry out the research, and the physicians and surgeons for allowing me to investigate cases under their care. In particular, I desire to express my gratitude to my chief, Sir James Walton, for his generous encouragement and for the help and advice which he has constantly given. My thanks are also due to the artist, Miss McLarty, for her patience and skill and for the care with which she has prepared the paintings.

FUNNEL-NECK DEFORMITY OF THE BLADDER

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INTRODUCTION

SINCE the days of Romberg disturbance of micturition has been recognized as one of the commonest symptoms of lesions of the spinal cord, and of tabes dorsalis in particular. Gowers has pointed out " that it may be only for these that the patient seeks advice." Following his introduction of the modern cystoscope, Nitze described the presence of trabeculation of the bladder wall in tabes dorsalis. Since then there has been considerable discussion as to the characteristic vesical findings in disease of the central nervous system. Most reports on this subject have dealt with the importance of trabeculation. This trabeculation has by many been attributed to atony and atrophy of the bladder wall, of which the internal vesical sphincter is an integral part. It might have been foreseen, therefore, that the sphincter would partake in such changes, and it comes as somewhat of a surprise that until recently relatively few observations should have been made on the appearance of the vesical sphincter in nervous disease.

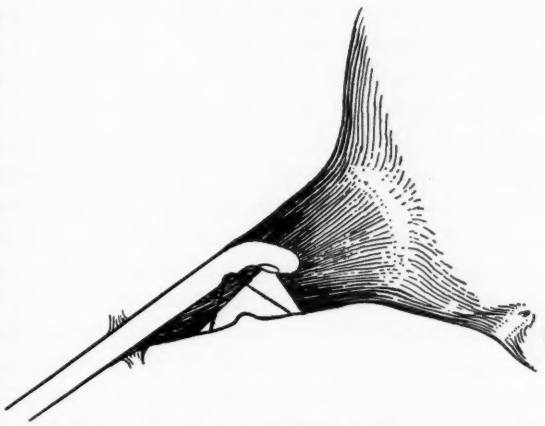

FIG. 360.—Pronounced funnel-neck deformity. Cystoscope held over verumontanum.

On cystoscopic examination of the normal male, if the instrument is withdrawn for a short distance from the bladder so that it lies within the posterior urethra, it is impossible to see the walls of that channel, as the urethral mucosa surrounded by the internal vesical sphincter is in close contact with both the prism and the lamp of the cystoscope. In certain circumstances, however, the internal urethral orifice is flaccid, and it is then possible to withdraw the cystoscope into the posterior urethra and to obtain a clear view of its postero-inferior wall and of the verumontanum (*Fig.* 360). This condition has been variously termed 'funnel-neck deformity of the bladder', or 'funnel-shaped posterior urethra'. From consideration of the bladder musculature it appears that such a condition might arise by dilatation of the prostatic urethra following either paralysis or paresis of the internal sphincter, the whole posterior urethra thus assuming a conical shape with the base of the cone directed towards the bladder

and the apex at the triangular ligament. This explanation may be provisionally accepted as the anatomical basis of the condition.

The funnel-neck bladder must not be confused with an appearance which has much in common with it but which arises from quite a different cause. In prostatic hypertrophy the enlarging adenoma frequently dilates the internal sphincter, and the growth of the lateral lobes produces a deep cleft-like urethra at the bottom of which, if the cystoscope is held at a suitable angle, the floor of the urethra together with the verumontanum can be clearly seen. A true funnel-neck deformity can exist only in the absence of prostatic enlargement.

REVIEW OF THE LITERATURE

In 1914 Alexeieff demonstrated to the Russian Urological Society a patient who from childhood had suffered from nocturnal enuresis, and in whom the posterior urethra with the colliculus seminalis was visible. Later in the same year he published his communication "On the Diagnosis of Nocturnal Enuresis." To Alexeieff the gaping posterior urethra indicated a persistent weakening of the internal sphincter, and offered an explanation of the enuresis.

In the same year, and prior to the above observation, Barrington demonstrated dilatation of the posterior urethra following experimental lesions of the nervous system. He obtained cystograms of etherized female cats, and under certain conditions (following division of the pelvic nerves, of the sacral dorsal roots, or transection of the spine in the lower thoracic region) observed some dilatation of the upper part of the urethra.

In an article entitled "Prostatisme sans Prostate" Randall (1915) wrote, "By gently withdrawing the instrument with the objective posterior and the ocular well depressed, it is possible to detect relaxation of the internal vesical sphincter, which is pathognomonic of spinal cord disease, especially tabes dorsalis. The picture thus obtained is at times remarkable, and often the entire floor of the posterior urethra may be observed with the Nitze cystoscope."

So far as we are aware this is the first reference to funnel-neck deformity in the American or English literature. Randall, however, in a private communication to Natanson, does not claim originality for the observation, but states that H. H. Young first drew his attention to the phenomenon about 1910. It seems improbable that the sign was well-known at this time, as Barney in his series of articles on the tabetic bladder in 1910 makes no reference to it either in his own patients or in his review of the literature. Koll (1915) and Greenberg (1916), in articles dealing with the cystoscopic appearances of the bladder in nervous lesions, also do not comment on the phenomenon.

In 1916 Caulk and Greditzer reported their observations on the cystoscopic appearances of the bladder in fifty cases of disease of the central nervous system. Their cases included examples of tabes dorsalis, dementia paralytica, post-apoplectic conditions, tumours of the cord, and other diseases. They comment on the visibility of the posterior urethra, and, although they have also observed this condition in chronic prostatitis and seminal vesiculitis, believe that the sign is of considerable diagnostic importance. They attempted to obtain cystograms showing the continuity of the bladder and prostatic urethra, but to their surprise found a well-defined boundary at the vesical orifice and no silver solution in the urethra. These

observers were confident that a diagnosis of tabes dorsalis and spinal cord disease may almost always be made by cystoscopic examination, and that frequently the bladder evidence is available before any other lesions are manifest.

In another paper (1919) Caulk and Greditzer, in conjunction with Barnes, reported further cystoscopic findings in 500 cases of nervous and mental disease. They confirmed their previous belief in the diagnostic importance of a funnel-neck bladder and described various degrees of relaxation. They found that in some instances only the first part of the urethra was visible, in others the whole prostatic urethra permitted indistinct inspection, whilst in a third group there was well-marked relaxation and the whole floor of the urethra and the verumontanum could be identified. These authors regard this latter degree as a characteristic and significant finding in organic nervous disease. The other types are less convincing, and seem to occur quite frequently in the presence of a negative neurological examination, as also in certain psychoses. In patients suffering from tabes dorsalis the sign was present in 80 per cent of cases. In the female, dilatation of the sphincter is more difficult to identify, but the authors believe that relaxation in the presence of trabeculation suggests a nervous lesion.

In 1917 Burns published his observations on bladder changes due to lesions of the central nervous system. Twenty of his twenty-one cases suffered from tabes dorsalis. He noted the diminution in tone of the bladder musculature, and found the vesical orifice to be either remarkably dilated or to exhibit considerable diminution in its tonicity. The cystoscope could be drawn down into the posterior urethra and the floor of that passage together with the verumontanum could be readily observed. Burns, like Caulk and Greditzer, endeavoured to show this dilatation by means of cystograms, but found that radiographic evidence was obtained only when complete incontinence existed. Loss of ureteral tone was occasionally shown by fluid flowing up the ureter. Burns furthermore resected the posterior sacral roots in dogs and demonstrated dilatation of the internal sphincter and a funnel-shaped posterior urethra, thus supporting Barrington's observations on cats.

Several other references appear in the American literature. Braasch (1917) considers dilatation of the internal sphincter a late phenomenon in nervous disease. In conjunction with Hager (1927) he points out that cystograms in patients suffering from cord lesions frequently portray three cardinal findings: (1) A large irregular bladder outline with trabeculation; (2) Relaxation of the internal sphincter; and (3) Regurgitation of the medium into one or both ureters.

In fourteen cases of shell fracture of the spine submitted to cystoscopy, Plaggemeyer (1919) noted normal or hypertonic contraction of the external sphincter but complete relaxation of the posterior urethra, so that the verumontanum appeared to lie in the floor of the bladder. In a series of three hundred and fifty cystoscopies Cumming (1922) noted the phenomenon twelve times. These patients suffered from a variety of urinary diseases, but none was proved to have a neurological lesion. The picture of the vesical neck was similar to that seen in a neurological bladder, except that the internal sphincter tended to retain some tone so that " there is a zone of poor focus " as the cystoscope is withdrawn over the sphincteric ridge. In these cases Cumming believed that the phenomenon did not exist except with the instrument in position. He, however, agrees that the phenomenon is usually well-marked in spinal lesions.

Meyers (1926) also refers to the funnel-shaped outlet of the tabetic bladder. He attributes the relaxation of the internal sphincter to the destructive effect of tabes on the sympathetic supply to the bladder, and likens it to paralysis of the ciliospinal fibres of the sympathetic causing the extreme myosis frequently observed in this disease.

More recently Learmonth, working on the neurophysiology of the urinary bladder in man, observed that whilst sacral anæsthesia caused some dilatation of the internal sphincter, the phenomenon became more pronounced after division of the sympathetic supply. Stimulation of the presacral nerve or its lateral roots caused contraction of the internal sphincter. The dilatation which followed division of the sympathetic was not, however, permanent, and Learmonth states that " the internal sphincter may close completely. However, it can be easily opened by the advancing beak of the cystoscope and gives one the impression that it opens more easily than normal." He concludes that the ability of the sphincter to remain closed after sympathetic neurectomy proves that it must itself possess inherent tone. He refers to the fact that in the ' cord-bladder ' the internal sphincter is found to be relaxed, and states that " in these cases the lesion is situated somewhere in the parasympathetic pathway. We are forced to conclude that maintenance of closure of the internal vesical sphincter depends upon its inherent tonus, which in turn depends upon the integrity of reflex fibres in the parasympathetic pathway; and that its sympathetic supply merely reinforces this closure."

In the British literature D'Arcy McCrea (1926) refers to funnel-neck deformity of the bladder, but found it only once in a series of thirty cases of tabes dorsalis. He also noted the phenomenon following sacral anæsthesia.

One of the present writers (J. B. M.) in an address to the Section of Urology of the Royal Society of Medicine (October, 1934) dealt amongst other things with funnel-neck deformity of the bladder, and the present investigation is a continuation and elaboration of certain work therein reported.

In a recent article on " Bladder Function in Spinal Injury ", Watkins reports the appearnace of funnel-neck deformity in lesions of the conus and cauda equina.

On the Continent considerable attention has been given in certain clinics to funnel-neck deformity. In the German literature, which of course includes many articles from Russian and Austrian sources, a fair number of references may be found. Carl Schramm in 1920 published a paper describing in detail six cases in which he had observed this phenomenon. He claimed originality for his observations, and was apparently unaware of the previous publications in both American and German literature. It is, however, noteworthy that the cystoscopic examination at which he first observed this phenomenon was carried out in January, 1912. His cases included two of spinal injuries, and one each of tabes dorsalis, disseminated sclerosis, spina bifida, and a case in which complete paresis of the bladder-closing mechanism was present but no cause could be found (' Kriegsdienstbeschädigung '). From this time the phenomenon has been referred to in the German literature as " Schrammsches Phänomen ". Schramm found in many of his cases, in addition to the paralysis of the internal sphincter, a flaccid paralysis of the floor of the pelvis, atony of the sphincter ani externus, and various sensory disturbances. He concludes that the sign may appear as an early symptom of affection of the central nervous system and will frequently do so when all other symptoms are still absent. The phenomenon is, in his opinion, objective and indisputable proof of permanent

damage to the bladder-closing mechanism. In some traumatic cases where function has been re-established by recovery of the sphincter externus, gaping of the internal sphincter persists and is recognizable evidence when other neurological signs have disappeared. Schramm was so confident as to the value of the sign that in cases of legal dispute he believed incontinence of urine may be discredited in the absence of objective findings at the bladder neck. A later communication makes it doubtful if Schramm still upholds this last conclusion. At the German Urological Congress of 1926 he also stressed the fact that the phenomenon is positive only if the colliculus appears at once on withdrawal of the cystoscope but without depression of the ocular.

Pfister was the first to question Schramm's views. He could establish no relationship between dysfunction of the sphincter and visibility of the posterior urethra, and denies the diagnostic value of the sign. He states that on many occasions when the colliculus was visible, a strong contraction of the sphincteric muscle subsequently occurred. He quotes six cases of spinal trauma in which the sign was present, but in a further six cases of definite vesical paralysis following injury to the cord the sign was absent. Three cases of the deformity in which examination of the central nervous system gave no pathological findings are also mentioned.

In 1922 Oswald Schwarz (Vienna) published a severe criticism of Schramm's views. Stating that he himself had observed a funnel-neck urethra in a case of tabes as early as 1920, he admits that the sign may occur, but denies to it any diagnostic significance. In proof of his assertion he points out that the phenomenon is frequently absent in cases of unmistakable nervous disease, and that even when observed it is extremely inconstant, being present on one examination and absent on another; indeed it may appear and disappear during a single cystoscopy. Schwarz, like Pfister, has observed the phenomenon when no lesion of the central nervous system could be demonstrated. He maintains that the longer the cystoscope remains in the bladder the more likely is the phenomenon to develop, and especially if the ocular end of the instrument is unsupported will its vertical drag cause a dilatation of the sphincter and the artificial production of the phenomenon. In support of these arguments he states that in his experience normal ejaculation (which depends on the internal sphincter for the forward direction of the sperm—*see* pp. 507, 513) may occur in those showing the phenomenon. The base of the bladder, as seen on a cystogram, also exhibits a normal outline. Oswald Schwarz maintains that the symptoms of disturbed bladder function point to hypertrophy of the internal sphincter rather than to its relaxation. The dilatation of the sphincter may be reflex rather than mechanical in origin, the irritation of the posterior urethra by the cystoscope possibly inducing Barrington's fourth reflex. He draws an analogy between the state of affairs occurring at the internal sphincter and the condition obtaining at the lower end of the œsophagus in cardiospasm, and points out that in the latter the passage of a sound produces relaxation of the sphincter.

Goldenberg (1924) confirmed Oswald Schwarz's views regarding the inconstancy of the sign and the possibility of its production by depression of the cystoscope. He also mentions maximal distension of the bladder and sacral anæsthesia as additional means of obtaining dilatation of the internal sphincter. In general he denies the value of the sign, but considers that it may be of some significance when combined with trabeculation and residual urine.

Against these adverse criticisms O. A. Schwarz (Berlin) and Perlmann appear as protagonists. They regard the sign as having great diagnostic value, and consider that it may appear as the only evidence of nervous disease. In one publication O. A. Schwarz quotes fifteen cases of tabes and cerebrospinal syphilis, in only one of which the sign was absent. He was particularly impressed by patients in whom, although there had been quite indefinite disturbance of micturition or none at all, the sign was found positive, and in whom subsequent careful neurological examination showed the existence of tabes. Schwarz also observed patients with a visible colliculus who were suffering from neurasthenia and in whom there was no demonstrable organic lesion. He expresses no view regarding the occurrence of the phenomenon in the healthy individual.

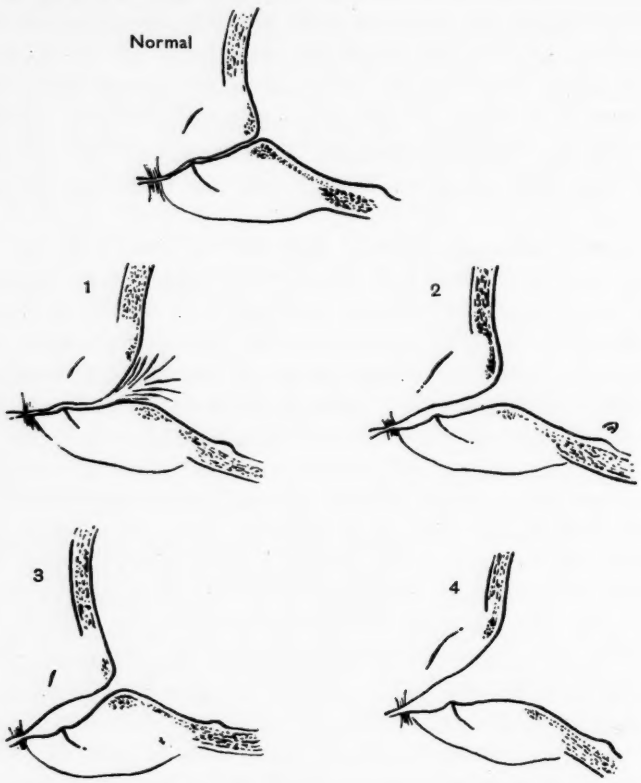

FIG. 361.—Types of deformity—Moro's classification. (*See* text.)

In view of these contradictory opinions Moro in 1927 investigated the material in his clinic and scrutinized the relationship existing between the trigone and posterior urethra in all cystoscopies. He differentiated four grades or degrees of dilatation (*Fig.* 361):—

Type I.—Radial streaking in the region of the internal urethral orifice, with more gradual transition of the bladder into the posterior urethra replacing the normal sharp sphincteric margin.

Type II.—The floor and side walls of the posterior urethra at the lower end are just visible and the colliculus is indistinctly seen as it lies close up against the prism.

Type III.—The field of view is dark in its proximal part, but as the cystoscope is further withdrawn it enters a relatively spacious, spindle-shaped chamber in which the illumination is bright. On the floor of this chamber the colliculus is seen, together with its surrounding valleculum.

Type IV.—The colliculus lies in full view in a brightly-lit channel, which allows some movement of the instrument to either side. The passage between the posterior urethra and the bladder is so wide that the posterior urethra appears as a funnel and the normal angulation at the point of transition fails (*see Fig.* 360, p. 501).

Moro placed diagnostic value only on *Types III* and *IV*. He did not invariably find the sign either in tabes or trauma. His conclusion was that when the nerve-supply of the bladder is damaged, gaping of the posterior urethra with visibility of the colliculus may occur. Once developed the phenomenon is constant, and may in traumatic cases persist as the only objective sign of preceding nervous injury. The sign is not, however, pathognomonic of nervous disease, as it may be artificially produced by tiring the sphincter, and thus may appear in healthy men. In such circumstances the sign is inconstant and may come and go during one and the same examination. As the sign, however, may indicate incipient nervous disease, it calls for a complete neurological overhaul.

In the same year (1927) Natanson also published his findings on the phenomenon, and he summarizes his views by stating: "It is certain that it occurs in patients with a healthy spinal cord, especially neurasthenics, but it is found so frequently in spinal and cerebral diseases (almost regularly in tabes and enuresis) that this association cannot be disregarded." He also admits that the establishment of a 'colliculus phenomenon' may, especially in tabes, be the first objective sign of existing disease of the spinal cord, and, in view of the importance of an early diagnosis, he sees no sufficient reason to deny the practical importance of the phenomenon.

In 1929 Fessler and Fuchs (Vienna) investigated sexual function in relation to funnel-neck deformity of the bladder. They pointed out that whilst in the normal male some of the secretion expressed by prostatic massage flows down the urethra and appears at the external meatus, the remainder runs back into the bladder and is subsequently voided in the urine. The proportion entering the bladder depends on the tone of the internal sphincter, and is maximal when this structure is paralysed. Five tabetics, in whom no secretion appeared at the external orifice following prostatic massage, exhibited the phenomenon on cystoscopic examination. Fessler and Fuchs also investigated fourteen male tabetics who, despite the well-known deterioration occurring in this disease, were still capable of sexual intercourse. Three gave a clear history that, although intercourse and orgasm were normal, there was a complete absence of ejaculation, the flow of semen undoubtedly passing back into the bladder.

The Russian literature contains several important papers on funnel-neck deformity of the bladder. The largest series so far examined is that of Epstein, Juschelevsky, and Michelson, who announced their results to the Russian Urological Association in 1928. They examined five hundred patients, two hundred of whom were known to be suffering from nervous disease; the remaining three hundred constituted the ordinary cystoscopic material of their clinic. Of the healthy men examined the vast majority showed no funnel-neck deformity. Attempts to produce the sign by movements of the cystoscope were also negative. The investigation of

the first group—namely, patients known to be suffering from disease of the central nervous system—appears to have definitely established that a funnel-neck depends not so much on the variety of the disease as upon its situation. The majority of cases showing it in well-marked development were instances of lesions of the spinal cord. In male patients suffering from tabes the sign was present in 90 per cent of cases, in syphilitic myelitis in 80 per cent. Examples of spina bifida, though not occurring in any numbers among their cases, were found to exhibit a high proportion of funnel-necks, whilst quite a few patients in whom the sign was positive, and who yet showed no central nervous disease, were found by radiology to have some degree of spina bifida which was otherwise unrecognizable. These authors conclude that a funnel-neck urethra is an early sign of nervous disease, and that its presence indicates an organic lesion or developmental defect. They also hold it to be unlikely that a funnel-neck is ever discovered in the healthy male, and in their opinion it is doubtful whether it is to be found in uncomplicated functional disturbance. These authors believe that dilatation of the internal sphincter may also be observed in women, although the phenomenon is never so pronounced as in the male. Their series of patients with nervous diseases included fifty women.

Halperstein (1928) found the sign on twenty-two occasions (tabes 12, spinal trauma 3, spina bifida 2, etc.) and attributed great value to it.

In another Russian publication, Dunajewsky and Michejew (1931) allow little diagnostic value to this sign, and have found it positive in a much smaller number of cases. Examining forty-two cases of tabes they found it present in but 16·6 per cent. Their observations on two hundred men, in whom there was no evidence of organic nervous disease and in five of whom the sign was present, led them to the conclusion that the sign could be artificially produced by the pressure of the cystoscope, and that it might be observed in healthy or neurasthenic patients. They considered trabeculation to be a more frequent and definite sign of nervous disease.

Recently Franz Gáspár (Budapest) has drawn attention to the fact that by using local anæsthesia it is possible to obtain complete relaxation of the internal vesical sphincter and to carry out with a cystoscope a complete inspection of the posterior urethra. He maintains that a better view is thus obtained than by using a posterior urethroscope. A catheter is inserted into the bladder and then withdrawn until its tip lies between the internal and external vesical sphincters. A local anæsthetic is then slowly injected over a period of half an hour, and a further half-hour is allowed to elapse before cystoscopy. Using novocain (1 per cent) or percaine (1–1000) full dilatation of the posterior urethra was obtained in eighteen out of twenty cases.

Short references to funnel-neck deformity of the bladder have also been made by Gagstatter, Schwarzwald, Young, Heplar, von der Becke, and Deutsch.

CLINICAL OBSERVATIONS AND CYSTOSCOPY

From a survey of the literature it is evident that the significance of funnel-neck deformity as a sign of nervous disease is by no means decided. We have endeavoured, therefore, to obtain an independent view of the incidence and diagnostic importance of this sign. Our inquiry concerns :—

1. Patients definitely suffering from nervous disease.

2. The frequency of the sign amongst urological patients who exhibit no evidence of nervous disease.

In examining the vesical neck for the sign, the fenestra of the cystoscope is first directed towards the trigone of the bladder and is then gradually withdrawn into the urethra. During this manœuvre it is necessary to depress the ocular end of the cystoscope slightly, otherwise the condition may escape observation. Only the floor and the lower portions of the lateral walls of the urethra can be brought into view. If the instrument is rotated, the field of vision becomes dark as soon as the upper portion of the side wall is reached, and even by manœuvring the instrument we have never been able to get a satisfactory view of the upper portion of the urethra.

The sign has been regarded as positive only when it has been recognized immediately on withdrawal of the instrument and has remained constant during the examination. Straining to micturate produces a temporary physiological dilatation of the posterior urethra.

To standardize our observations and facilitate comparison, we have adopted the classification of Moro in stating our results (p. 506 and *Fig.* 361). This classification includes all recognizable degrees of the phenomenon. In practice there is difficulty in distinguishing with certainty *Types I* and *II* of this classification from the normal. Hence we, like Moro himself, regard these grades as unreliable, and although we have used them in classifying some of our material, we do not attach any clinical significance to them, and regard only *Types III* and *IV* as constituting a funnel-neck deformity.

I. OBSERVATIONS ON PATIENTS WITH NERVOUS DISEASE

Our material consisted of 17 cases of tabes dorsalis, 5 cases of general paralysis of the insane, 2 cases of cerebrospinal syphilis including one of Erb's syphilitic spinal paralysis, 3 cases of disseminated sclerosis, and 1 of 'spastic paraplegia'. These patients were not chosen on account of the existence of urinary symptoms, but represented such material as was available for examination. The only selection we allowed ourselves was the rejection of cases in which complications would tend to alter the appearance of the bladder-neck. Infection is one such complicating factor, and accordingly all who showed evidence of cystitis, either by a dirty urine or on cystoscopy, were rejected. In practice this did not involve the exclusion of any large number, as the infrequency of this condition, even in the presence of fairly large amounts of residual urine, has been surprising. Enlargements of the prostate were also excluded.

A viewing cystoscope was used for the examination, and, recognizing the susceptibility of the bladder to infection in central nervous disease and the importance of keeping it uninfected, the most rigid asepsis was maintained. No anæsthetic of any type was used. This allowed judgement of the degree of pathological anæsthesia, and entailed little or no hardship to the patients, as in most of them urethral sensation was either diminished or almost absent. The residual urine was measured in most instances, the average amount present being 250 c.c.

Examination of the Vesical Orifice.—This revealed a funnel-neck in a high proportion of our patients. Thus, of the 17 tabetics, only 3 showed a normal orifice. The examples of general paralysis and of cerebrospinal syphilis all showed the deformity. The sign was absent in those suffering from disseminated sclerosis and spastic paraplegia, except for one example in the former disease. In this patient it was necessary to use a local anæsthetic (percaine), and this result must therefore be accepted with scepticism.

The bladder was filled in the first instance with 200 c.c. of fluid. With this amount, sagging of the vesical wall often showed that the bladder was obviously not distended and was incapable of contracting down on its contents. With such a degree of distension several cases did not exhibit a funnel-neck deformity, though when the bladder was filled to its individual capacity the sign was found fully developed. The filling was never carried to the point of causing discomfort, and in most patients much more fluid might have been introduced. This fact accounts for the occurrence of two figures attached to several of the cases in our own table of results (*Table I*, p. 512). The first figure indicates the appearance when using 200 c.c. of fluid, the second that with the bladder more fully distended. The dilatation of the posterior urethra was extremely wide in many, the trigone appearing to merge with the floor of the prostatic urethra without the intervention of a dividing rim. Excellent views were obtained of the urethral crest terminating in the colliculus, the latter projecting from the floor of the urethra as a pale pink hummock bounded on either side by the valleculum. The openings of the prostatic utricle and of the ejaculatory ducts were often recognized on the surface of this structure. Occasionally the urethra was dilated for a short distance below the colliculus. Amongst patients exhibiting *Type III* of the deformity, the dilatation of the mid-section of the prostatic urethra appeared as great as that observed in the more advanced grade, the difference between the two varieties being the constriction at the vesical orifice of the former.

The retrogression and final disappearance of the phenomenon following anti-syphilitic treatment, which Burns observed in his series, were not noted by us.

The Bladder Wall.—The general impression gained from observation of the bladder wall in the patients suffering from syphilitic disease of the nervous system was one of atony and atrophy. The vesical wall as a rule appeared extremely thin, and intestinal movements were observed with great frequency. Trabeculation was present in 15 cases. In 12 of these it exhibited the characteristic fine and delicate appearance described by Thomson-Walker. In the other 3 bladders the degree of trabeculation was either insignificant or atypical. The ureteric and inter-ureteric bars frequently lacked their normal fleshiness, and as the large retro-trigonal recess fell away steeply behind them, they appeared to be unduly projected into the bladder.

Cystography.—Many attempts were made to obtain cystograms showing the funnel-neck deformity. With four exceptions (*Cases* 2, 7, 10, 22), however, these efforts ended in failure, the bladder showing the normal smooth basal contour, with no opaque solution in the posterior urethra. Irregularity in the shape of the bladder and the coarser varieties of trabeculation are shown by this means. Twice the opaque fluid regurgitated up one or other ureter.

The relative infrequency of cystograms showing a funnel-neck deformity seems remarkable, but is in accordance with the findings of other observers (Caulk and Greditzer, Otto Schwarz, Burns). The last-mentioned author obtained outlines of the prostatic urethra only when complete incontinence existed.

The technique of cystography consisted in filling the bladder with the opaque solution through a catheter, which was withdrawn prior to exposure of the film. The volume of fluid employed depended on the previously determined bladder capacity. Potassium iodide (2·5 per cent) and silver iodide (5 per cent emulsion) were both tried as opaque solutions, the latter being reputed to be less irritant than

the former. Each of these solutions, however, was liable to cause discomfort and a desire to micturate. Efforts to maintain the bladder pressure at a low level were thus frequently frustrated, and whilst in some instances large volumes of fluid were introduced at a pressure below 10 cm. of water, in others small volumes (200 to 250 c.c.) caused discomfort, and the bladder pressure, despite a period allowed for the establishment of equilibrium following filling, remained at 30 to 40 cm. of water.

Guyon, as long ago as 1901, showed that following section of the nervi erigentes and injection of an irritant ($AgNO_3$) the bladder would still contract and expel part of its contents, and from this concluded that under certain conditions the bladder musculature was independent of all medullary influence. Head and Riddoch have described complete automatic micturition, consisting not only of contraction of the bladder but of relaxation of the sphincter, following destruction of the sacral roots by trauma. Denny-Brown and Graeme Robertson also believe that automatic micturition may occur following complete sacral lesions, and that the internal sphincter relaxes reciprocally with contraction of the detrusor.

These observations suggest that an artificially produced funnel-neck deformity might be shown on cystograms obtained with an irritant solution, either by the occurrence of contraction of the detrusor and reciprocal relaxation of the internal sphincter, or by contraction of the detrusor raising the intravesical pressure sufficiently to force the internal sphincter. Despite this, in the above series, the deformity was but rarely found on cystograms. Even when a positive cystogram is obtained it must be determined whether it faithfully depicts an existing condition or is artificially produced. In illustration of this view *Case* 22 may be cited. Cystography performed on this man with a potassium iodide solution showed a large funnel-neck deformity (*see Figs.* 364–367, pp. 522, 523), whereas subsequent excretion cystography failed to demonstrate any deformity of the bladder neck. The relatively high intravesical pressures recorded in some instances prior to the withdrawal of the catheter and the absence of opaque fluid in the posterior urethra on subsequent cystograms also throw doubt on the pressure usually stated to be sufficient to force the internal sphincter.

It thus appears that whilst ascending cystography may satisfactorily serve most ordinary purposes it is unsuitable for observations on the bladder neck. So many complicating factors are introduced that unless the bladder pressure has been low prior to the withdrawal of the catheter little value can be placed on the evidence. Excretion cystography offers material advantages, as the fluid is less irritating and mechanical interference with the posterior urethra is avoided.

Relationship between Urinary Symptoms and Funnel-neck Deformity.—The order of onset of urinary disturbance in tabetics is variable, but in our experience the following would appear to be the most usual course: Diminution or loss of the normal desire to micturate leads to infrequency of the act, urination taking place only once or twice daily. The quantities passed are then necessarily large. Coincidently the patients may experience difficulty in starting the act and the stream may lack force and be intermittent. The whole of the vesical contents are not expelled and residual urine commences to collect. Signs of incontinence may appear at this stage, or may have shown themselves at an earlier time; in some instances they constitute the initial symptom. At first incontinence may be limited to occasional nocturnal enuresis, or dribbling following micturition. Sudden

exertion may cause the escape of small jets of urine. With progression of the disease the volume of residual urine tends to increase, and abdominal straining becomes more and more necessary to empty the bladder. The incontinence also may become more pronounced, a distended bladder being frequently accompanied by either intermittent or almost continuous dribbling of urine. Active incontinence was not observed amongst the tabetics, but occurred once in a case of spinal syphilis and again in a single instance amongst the general paralytics. No enormously distended bladders occurred in any of our patients.

Difficulty in micturition, though frequently present in minor degree, has not been a troublesome symptom, and complete retention has occurred in the early stages but once. One other patient complained of attacks of acute retention, but emergency catheterization never discovered more than three or four ounces of urine in his bladder. There have been no instances of urethral anæsthesia so complete that micturition has been recognized only by wetting of the clothes.

Table I.—FUNNEL-NECK DEFORMITY IN NERVOUS DISEASE

DISEASE	PRINCIPAL URINARY SYMPTOM	CASE AND DEGREE OF DEFORMITY
1. Tabes dorsalis (17)	1. No definite symptoms (3) ..	*Case* 2 (*IV*); *Case* 3 (*IV*); *Case* 9 (0 → *IV*)
	2. Absence of desire to urinate (3). Infrequent micturition with passage of large amounts of urine	*Case* 1 (*I* → *IV*); *Case* 4 (*II* → *IV*); *Case* 16 (*IV*)
	3. Incontinence, dribbling, nocturnal enuresis (8)	*Case* 5 (*III* or *IV* → *IV*); *Case* 6 (*III*); *Case* 7 (*IV*); *Case* 8 (*I or II*); *Case* 10 (*II* → *IV*); *Case* 12 (*IV*); *Case* 14 (*IV*); *Case* 17 (*II* → *IV*)
	4. Difficulty (2)	*Case* 13 (*II* → *IV*); *Case* 15 (0)
	5. Sensation of distension without residual urine (1)	*Case* 11 (0)
2. G.P.I. (5) ..	1. No symptoms (2)	*Case* 19 (*III*); *Case* 20 (*II* or *IV*)
	2. Dribbling urine (2)	*Case* 18 (*III* → *IV*); *Case* 22 (*IV*)
	3. Active incontinence (1) ..	*Case* 21 (*IV*)
3. Cerebrospinal syphilis (2)	Active incontinence	*Case* 23 (*IV*)
	Incontinence	*Case* 24 (*III*)
4. Disseminated sclerosis (3)	——	(0); (0); (*III*)
5. Spastic paraplegia (1)	——	(0)

Table I shows that there is no real difference in the incidence of funnel-neck deformity amongst those complaining of incontinence and those who have no such trouble. The internal sphincter normally retains the urine within the

bladder and is possibly responsible for continence during sleep, the external sphincter relaxing as do all voluntary muscles. It might therefore have been anticipated that paresis of the internal sphincter would play a part in the nocturnal incontinence so often seen in tabes. On the other hand, the internal sphincter is usually destroyed or out of action following the operation of prostatectomy, and yet these patients are not incontinent at night. Likewise the internal sphincter may be destroyed by trauma and yet continence be complete.* The external sphincter in the normal male is therefore quite capable of assuming this function of the internal sphincter. The onset of incontinence in tabes dorsalis thus suggests not only that the internal sphincter is atonic, but that the external sphincter is undergoing similar changes. The discovery of a funnel-neck deformity does not necessarily predict the onset of incontinence.

Sexual Function.—Realizing that the internal sphincter is the barrier which prevents sperm passing back into the bladder during ejaculation and that weakness of this muscle might lead to a failure of this function, we inquired in a few cases regarding emission. It was not, however, possible to investigate the sexual function of all patients in this series. Of the tabetics, 2 reported that orgasm without ejaculation occurred, whilst on the other hand 2 others who exhibited the deformity were emphatic that a normal emission of semen occurred.

Discussion

From the above observations it is evident that in syphilitic disease of the central nervous system the cystoscope will almost invariably reveal a funnel-neck deformity of the bladder. The most reasonable explanation of the phenomenon is weakness of the internal vesical sphincter. The appearance suggests that the bladder and dilated posterior urethra form one cavity, but cystography in the majority of instances refutes this suggestion, and establishes beyond reasonable doubt that the deformity usually exists only when the cystoscope is in position, and the question therefore arises whether it may not be artificially produced. Depression of the cystoscope has been advanced as a possible cause, but endeavours to produce the phenomenon by this means in the healthy male with a normal posterior urethra have met with little or no success, and even the steepest depression has never produced a *Type III* or *IV* deformity. The cystoscope alone, therefore, is insufficient to produce the phenomenon, and the sign must primarily indicate weakness of the internal sphincter.

Conversely, the absence of a resultant shadow on cystography shows that the internal meatus is closed when no instrument occupies it, and the dilatation seen cystoscopically must be due to mechanical separation of the weakened sphincter. This closure so frequently observed on cystograms may be due on the one hand to tone remaining in the internal sphincter sufficient to close the urethra, or on the other hand to elastic tissue, of which an abundance is known to exist in this situation, performing the task. Whichever explanation is true, and it seems probable that a combination of forces operates, the closure of the bladder neck must be quite weak, as the lightest movement of the cystoscope suffices to separate the urethral walls in a manner usually impossible in the normal unanæsthetized male urethra. That elasticity of the tissues around the bladder neck may act in this way

* See two cases reported by one of us (J. B. M.), *Proc. Roy. Soc. Med.*, 1934, xxviii, 1.

is suggested by the fact that in complete incontinence due to paralysis of the internal and external sphincters the outflow of the urine is not continuous. Small quantities collect during periods of inactivity and are expelled only when movement temporarily raises the intravesical pressure. It is also well known that urine does not escape from the bladder following death (no matter what the position of the body). At the Urological Congress of 1933 Weijtlandt quoted Guthrie (1834) and Barkow as regarding the sphincter not as a muscle but as elastic tissue, whilst Kenneth Walker also drew attention to the importance of elastic tissue in this situation. Watkins quotes Schwarz and Dennig as having made similar observations.

The nerve-supply of the bladder is derived from the sympathetic and sacral parasympathetic system. Much diversity of opinion exists concerning the exact distribution and function of each system. In man, according to Learmonth, the sympathetic supply arises from the first, second, third, and fourth lumbar ganglia on either side, and the intermesenteric plexus, and proceeds to the bladder by the presacral and hypogastric nerves. The segmental level of the connector cells is not known with accuracy, but is frequently attributed to the first and second lumbar segments. The afferent sympathetic fibres enter the spinal cord between the ninth thoracic and fourth lumbar segments. The sacral autonomic fibres (afferent and efferent) arise from the second and third sacral segments. The prostatic urethra and external sphincter are supplied by the pudendal nerve, which springs from the third and fourth sacral segments. For the purpose of this discussion it is only necessary to consider the function of the sensory paths. It is probable that the afferent sympathetic fibres convey painful impulses and also some information regarding distension. The main sensory path is, however, the parasympathetic system. The degree of distension of the bladder, tactile and thermal stimuli, and some painful impulses are conveyed by this route. Its fibres also include the vast majority of those concerned with the micturition reflexes, and a proportion of sensory fibres from the posterior urethra.

The primary lesion in tabes is a syphilitic inflammation of the posterior nerve-roots central to the ganglion, with subsequent degeneration of the efferent neurons and sclerosis (Richter). These changes appear first in the lower lumbar and upper sacral regions. In time complete atrophy of the ganglion cells and of the peripheral portions of the sensory nerves may occur. The afferent autonomic fibres are affected in this degeneration. The efferent fibres and ganglion cells probably escape.

Since the tabetic lesion commences in the lumbosacral region the main sensory tracts from the bladder are impaired at an early date. Though gradually losing its sensory supply, the bladder remains in contact with the motor centres of the brain and spinal cord. The hypogastric ganglia and vesical plexuses are also probably unaffected. The tabetic bladder, therefore, differs in important respects from the ' cord-bladder ' described by Head and Riddoch, Holmes, Denny-Brown and Robertson, etc. In the cord-bladders described by these authors there are two main groups. In the one a transverse lesion of the cord leaves the sacral reflexes complete; in the other a conus or cauda equina lesion destroys the sacral connections, but its sympathetic supply and its connections with the hypogastric and vesical ganglia remain intact.

In our own experience the relaxation of the internal vesical sphincter occurring in tabes appears to be part of a general loss of tone of the bladder musculature. The collection of residual urine, the incompetence of the vesical sphincters, and

the atrophic appearance of the vesical wall all suggest atrophy. Many observers (Frankl-Hochwort, Böhme, etc.) have ascribed the trabeculation to hypertrophy. Our own view agrees with that of Thomson-Walker, who attributes the characteristic appearance to atrophy of the bladder wall. The tone of smooth muscle is known to be much more independent of nervous reflexes than that of striated muscle, but the gradual and progressive loss of detrusor and sphincteric tone occurring in tabes suggests that the reflex arc is of considerable importance. It appears probable that the first result of the loss of proprioceptive impulses is a diminution of detrusor tone, which, apart from any loss of conscious sensation, is itself sufficient to account for the early onset of too infrequent micturition. Concurrently atonia of the internal sphincter develops and a funnel-neck deformity becomes visible on cystoscopic examination. The loss of tone in the detrusor and sphincters would appear to be progressive, the bladder, if uncomplicated by cystitis, tending to become a flaccid organ. Loss of the local sphincteric reflexes may cause some sphincteric difficulties, but except for the attacks of acute retention occurring at the onset of the disease (at which stage the heightened skin reflexes suggest the possibility of sphincteric spasm) there does not seem to be any evidence for assuming abnormal spasticity of the sphincters as a cause of retention. Whether retention or incontinence shall predominate depends on the relative atonicity of the detrusors and sphincters.

The results of section of the posterior sacral nerve-roots in animals provide an interesting comparison. In the dog, Dennig and Burns have shown the resulting disturbances of micturition to be almost identical with those of the advanced tabetic. Barrington, however, in similar experiments on cats, observed relaxation of the bladder with retention of urine, which if unrelieved proceeded to overflow incontinence. Efforts to express urine from the bladder in these cats were met by strong and sustained resistance. Cystograms showed the internal sphincter dilated and the fluid held at the external sphincter.

Case Reports of Nervous Disease

Case 1.—W. P. Aged 47. Tabes dorsalis.

Primary syphilis twenty-six years ago. No immediate treatment. Diagnosed as tabes dorsalis eight months ago. History of 'tiredness' but no other symptoms. Pupils now pin-point and show no reaction to light. Knee- and ankle-jerks absent. No loss of sensation. Wassermann and Kahn tests positive. Received eight months' treatment for neurosyphilis prior to examination.

Urinary Symptoms.—Passes large quantities of urine. Normal desire to micturate; good stream; no interruption. F.=D.2–3/N.0.* No retention. No incontinence. No enuresis. No intercourse with wife for nine months. No emission of semen.

Cystoscopy.—Passed 1320 c.c. urine prior to examination. Residual urine 200 c.c.; clear. Bladder filled with 200 c.c. fluid. Bladder wall in folds, obviously not distended. No cystitis. Trabeculation, especially in the roof of the bladder, very fine in type. Intestinal movements observed through the bladder wall. Bladder neck showed questionable degree of dilatation (*Type I*). Bladder further distended but without causation of any discomfort. Funnel-neck urethra developed (*Type IV*).

Case 2.—F. H. Aged 60. Tabes dorsalis.

Date of infection with primary syphilis unknown. Has suffered from upper abdominal pain for three to four years. Repeated X-ray examination has revealed no pathological lesion of the intestinal tract. Diagnosed as tabes dorsalis six months ago. Wassermann and

* For simplicity of recording frequency of micturition the above abbreviation has been adopted. Thus in the present example F.=D.2–3/N.0 indicates that micturition has occurred two to three times during the day and not at all at night.

Kahn tests positive (+ +). Had received six months' treatment for neurosyphilis prior to examination.

URINARY SYMPTOMS.—No urinary symptoms. Normal desire to micturate. No dribbling. No enuresis.

CYSTOSCOPY.—Urethra almost anæsthetic. Residual urine 30 c.c.; clear. Bladder filled with 200 c.c. fluid. No cystitis present. Beautiful fine and coarse trabeculation. Fully developed funnel-neck deformity (*Type IV*). Colliculus well seen in centre of a large illuminated cave. Urethra also considerably dilated below colliculus.

CYSTOGRAPHY.*—Cystogram shows a small bladder with well-marked trabeculation. The funnel-neck deformity is also shown. Some opaque fluid regurgitated up right ureter.

Case 3.—R. G. Aged 42. Tabes dorsalis.

Gonorrhœa nineteen years ago. Complaining of lightning pains for past five years. Pupils small, unequal, and fixed. Knee- and ankle-jerks absent. No loss of sensation over limbs. Blood Wassermann test positive + +. Cerebrospinal fluid shows 5 lymphocytes per c.mm.; Wassermann test +; Lange's colloidal gold curve 5 5 5 5 4 3 2 1 0 0 0.

URINARY SYMPTOMS.—Normal desire to micturate. F.=D.2–3/N.0–1. No difficulty. Never retention. No dribbling. No nocturnal enuresis. Married; normal emission.

CYSTOSCOPY.—Residual urine 15 c.c. Bladder distended to 200 c.c. Mucosa normal; no trabeculation. Well-marked funnel-neck deformity of bladder (*Type IV*). Colliculus and prostatic utricle well seen; urethra dilated for an inch below this. Bladder refilled with 400 c.c. fluid. No discomfort. No further dilatation.

CYSTOGRAPHY.—The cystogram (*Fig.* 362) shows trabeculation and a funnel-neck.

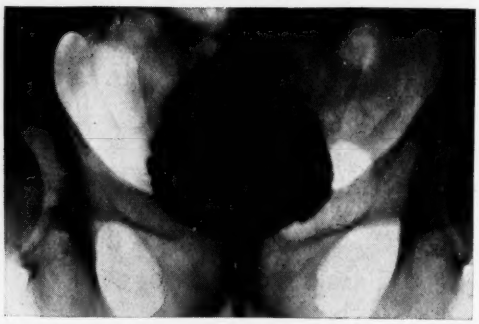

FIG. 362.—*Case* 3. Tabes. Cystogram showing well-marked trabeculation and a funnel-neck deformity.

Case 4.—T. J. L. Aged 58. Tabes dorsalis.

Contracted primary syphilis thirty-nine years ago in Hong Kong. Received mercurial treatment. During 1916 was invalided out of the Navy suffering from tabes dorsalis. Since then has been troubled with lightning pains and ataxia, but otherwise has remained well. At present his pupils are small, equal, and pin-point. The knee- and ankle-jerks are absent. Sensation is diminished over the legs and saddle area.

URINARY SYMPTOMS.—Infrequent micturition; F.=D.3–4/N.0. Desire somewhat diminished. No retention. No dribbling. Nine years ago used to wet his clothes, but never now. No nocturnal enuresis.

CYSTOSCOPY.—Residual urine 15 c.c. Urethra almost anæsthetic. Bladder filled with 200 c.c. fluid. No cystitis. Fair degree of trabeculation; coarse rather than fine. Internal urethral orifice appeared patulous (*Type II*). Bladder distended to 650 c.c. fluid without discomfort. Funnel-neck urethra (*Type IV*) developed. Good view of colliculus.

Case 5.—A. B. Aged 31. Tabes dorsalis.

Primary syphilis fifteen years ago. No immediate treatment. Two years ago sought medical aid on account of shooting pains in his thighs and weakness of right leg. Diagnosis

* Cystograms have been made in most cases, but were generally negative. A note has been made only where a funnel-neck was observed.

of tabes dorsalis established. Wassermann and Kahn test + +. Cerebrospinal fluid showed 10 cells per c.mm.; Wassermann test +; Kahn test + +; Lange's colloidal gold test 0012332100 0. At present shows marked ataxia. Pupils dilated; no reaction to light. Knee- and ankle-jerks absent. Sensation markedly diminished over legs and saddle area.

URINARY SYMPTOMS.—Retains his urine for an unusual length of time, then passes a large amount. No difficulty. No straining. Good stream. F.=D.4/N.0. May dribble a little after urinating. No enuresis.

CYSTOSCOPY.—Penile hypospadias. Urethral and bladder sensibility practically absent. Residual urine 240 c.c. Bladder filled with 200 c.c. fluid. No cystitis. The internal urethral orifice appeared patulous. This formed a loose waist between the bladder and a widely dilated prostatic urethra, in which the colliculus was clearly seen (*Type III* or *IV*). The bladder was then distended to 600 c.c. The funnel-neck deformity became more prominent (*Type IV*).

Case 6. P. B. Aged 56. Tabes dorsalis.

Date of primary syphilis unknown. Sought medical treatment fifteen months ago on account of pain in left side and dribbling of urine. Diagnosed as a case of early tabes dorsalis. Wassermann test (blood) positive (+ +). Pupils now small; no reaction to light. Knee-jerks present, ankle-jerks absent. Sensation is poor over the legs and perineum, especially over saddle area. Feels a pin but unable to distinguish the point from the head. Patient has now received a year's antisyphilitic treatment.

URINARY SYMPTOMS.—Three years ago, for a period of three to four months, suffered from nocturnal enuresis and slight dribbling incontinence during day. One year ago complained of dribbling incontinence but states that this symptom no longer troubles him. At present feels a desire to micturate, but does not think the sensation is as acute as previously. At times experiences a little difficulty in commencing micturition. Good stream; no interruption. Never retention.

CYSTOSCOPY.—Urethral sensibility very slightly impaired; feels pull on ligaments. Distension usual 200 c.c. No cystitis. Very many extremely large blood-vessels. Very marked trabeculation present, which, excepting the trigonal area, extended over the whole of the bladder including the apex. At first the bladder neck appeared normal, but on withdrawing the cystoscope slightly an illuminated cavern was entered, in which the colliculus was well displayed (*Type III*).

Case 7.—J. B. Aged 52. Tabes dorsalis.

Primary syphilis eleven years ago. Treatment for one week. Attended urological clinic complaining of difficulty in micturition. 'Shooting pains' down back of thighs for several years. Ataxic gait. Romberg's sign positive. Pupils small; no reaction to light. Knee- and

FIG. 363.—*Case* 7. Tabes. Cystogram showing bladder half filled with potassium iodide solution. Pressure prior to withdrawal of catheter was below 5 cm. of water. Large smooth bladder showing funnel-neck deformity and regurgitation of fluid up left ureter.

ankle-jerks absent. No real loss of sensation demonstrated. Wassermann and Kahn tests (blood) positive (+ +). Cerebrospinal fluid: Wassermann —; Kahn ±; Meinicke ±.

URINARY SYMPTOMS.—Difficulty of micturition. Strains to urinate, so much so that his hæmorrhoids bleed. Passes urine infrequently; F.=D.2–3/N.0–2. At times does not feel his urine flowing. Has to wear a cloth to save his clothes.

CYSTOSCOPY.—Urethral sensation diminished. Residual urine 480 c.c. Bladder filled with 200 c.c. fluid. Obviously not distended. No cystitis. Beautiful fine trabeculation. Mucosa has an atrophic appearance. Funnel-neck urethra (*Type IV*). Excellent view of verumontanum. Bladder filled with 800 c.c. fluid. No discomfort whatever. Fine trabeculation and atrophic appearance of mucosa very marked. Funnel-neck urethra unchanged.

CYSTOGRAPHY.—Cystogram (400 c.c.) showed no trabeculation; funnel-neck demonstrated (*Fig.* 363). Some opaque fluid escaped up L. ureter. With 600 c.c. the vesical pressure was still below 5 cm. water.

Case 8.—W. S. Aged 58. Tabes dorsalis.

Date of primary syphilis unknown. Four years ago a left orchidectomy was performed for gumma of left testis. A diagnosis of tabes dorsalis was also made at this time. Patient subsequently underwent a course of antisyphilitic treatment, but attended irregularly. Condition now very much worse. Walks with great difficulty; tabetic gait. Romberg's sign well-marked. Pupils unequal and do not react to light. Knee-jerks absent. Sensation diminished from waist down; loss almost complete in saddle area.

URINARY SYMPTOMS.—Nocturnal enuresis for three to six months. Can pass urine voluntarily but occasionally dribbles slightly. No retention. No frequency. Double incontinence of urine and fæces first noticed three months ago during sleep.

CYSTOSCOPY.—Residual urine 300 c.c. Urethra almost anæsthetic. Bladder distended with 200 c.c. fluid. Bladder mucosa injected. Some trabeculation of bladder; not fine type usually described as typical. Internal urethral orifice somewhat patulous (*Type I* or *II*). No well-marked funnel-neck deformity.

Case 9.—J. A. N. Aged 58. Tabes dorsalis.

Date of primary syphilis unknown. "Unable to control legs" for nine years. Ataxic gait. Romberg's sign strongly positive. Pupils pin-point and equal; no reaction to light. Knee-jerks absent. Lower half of body almost completely anæsthetic. Wassermann reaction strongly positive (+ +).

URINARY SYMPTOMS.—Subnormal desire to micturate. No retention. No dribbling. No enuresis.

CYSTOSCOPY.—Residual urine 300 c.c. Urethra nearly anæsthetic. Bladder distended with 200 c.c. No cystitis. Slight trabeculation. No funnel-neck deformity. Bladder then filled to 1000 c.c. fluid; no discomfort. Well-marked funnel-neck deformity then observed (*Type IV*).

Case 10.—R. D. Aged 60. Tabes dorsalis.

Primary syphilis over thirty years ago. No immediate treatment. "Gastritis" nine years ago; at this time received nine months' treatment for syphilis. Diagnosed as tabes dorsalis ten months ago. Complains of weakness of legs for past two months. Sciatica in thighs and calves for several years. Pupils: very sluggish reaction to light. Knee- and ankle-jerks absent. No areas of anæsthesia detected. Wassermann test (blood) positive. Has received approximately ten months' treatment for neurosyphilis.

URINARY SYMPTOMS.—Infrequent micturition and passage of large quantities of urine. Subnormal desire to micturate. F.=D.2–3/N.0. Good stream, but interrupted at times. Occasionally urine dribbles away into his clothes. Nocturnal enuresis on a few occasions. No retention.

CYSTOSCOPY.—Residual urine 120 c.c. Urethral sensation apparently much diminished. Bladder filled with 200 c.c. fluid. Fine trabeculation of bladder wall. No cystitis. Slight degree of funnel-neck urethra (*Type II*). Bladder filled to 400 c.c. Well-developed funnel-neck urethra (*Type IV*). When bladder was distended to 625 c.c. some mild discomfort was felt.

CYSTOGRAPHY.—Cystogram (500 c.c.) showed smooth regular bladder outline. Slight funnel-neck deformity.

Case 11.—W. M. Aged 61. Tabes dorsalis.

Twelve years ago, followed an alcoholic debauch, developed retention. Catheterized. No history of venereal disease. No evidence of stricture or prostatic enlargement. Knee-jerks normal at this time. Subsequently attended hospital at verying intervals complaining of retention of urine, but on examination bladder was never distended. Cystoscopy performed nine years ago. Bladder showed slight trabeculation and false diverticula; no cystitis. No observation was made on bladder neck. During past year patient's symptoms have been more pronounced. Attends hospital regularly complaining of retention, and stating that he has not passed urine for several days, but that a little dribbles away. On catheterization never more than 75 to 100 c.c. urine obtained. No. 24 F. bougie passed easily. Eight months ago sprained left ankle, and since then a typical Charcot's joint has developed. Wassermann and Kahn reactions strongly positive.

CYSTOSCOPY.—Urethral sensation slightly diminished. Bladder distended with 200 c.c. fluid. No marked cystitis. Fine isolated trabeculations and false diverticula. No evidence of funnel-neck deformity. Slight prostatic hypertrophy.

Case 12.—A. R. Aged 51. Tabes dorsalis.

Date of primary syphilis unknown. Diagnosed as locomotor ataxia six months ago. History of "weary feelings" and headaches for previous two years. Mind obviously clouded. Pin-point pupils; no reaction to light. Knee- and ankle-jerks absent. Sensation over lower half of body markedly impaired. Wassermann test strongly positive. Received six months' treatment for neurosyphilis prior to this investigation.

URINARY SYMPTOMS.—"Can hold water for a long period." Stream interrupted. Believes he has finished and then has to start again. Occasional dribbling. No nocturnal enuresis.

CYSTOSCOPY.—Urethral sensation very diminished or absent. Urine clear. Bladder filled with 200 c.c. fluid. No cystitis present. Funnel-neck urethra (*Type IV*).

Case 13.—F. F. Aged 43. Tabes dorsalis.

Primary syphilis twenty years ago. Complained of pains in legs twelve years ago. Wassermann test positive at that time. Received antisyphilitic treatment for two years. Attended a Municipal Clinic six years ago. Diagnosed as a case of tabes dorsalis at this time. Has received treatment for neurosyphilis ever since. Two years ago developed Charcot's disease of left hip. Left knee-joint also disorganized. Now shows marked Romberg's sign. Pupils equal but react very sluggishly to light. Knee- and ankle-jerks absent. Some loss of sensation over whole body below waist line; very marked loss from knees down and over saddle area.

URINARY SYMPTOMS.—Difficulty in micturition for five to six years. Never complete retention. Normal desire to micturate. F.=D.3/N.0. Does not pass very large quantities. No dribbling. No enuresis.

CYSTOSCOPY.—Urine clear. Urethral sensation severely diminished. No residual urine. Bladder filled with 200 c.c. fluid. No cystitis. No trabeculation. Funnel-neck bladder (*Type II*). Bladder distended to 600 c.c.; slight discomfort. Funnel-neck more conspicuous (*Type IV*). Verumontanum well seen.

Case 14.—R. C. Aged 22. Tabes dorsalis (juvenile).

Congenital syphilis. Sought treatment three months ago on account of marked incoordination and difficulty in walking. Diagnosed as tabes dorsalis. Wassermann test (blood) positive. Cerebrospinal fluid showed greatly increased globulin; Wassermann test positive. Has now received three months' antisyphilitic treatment. His walking is stated to have improved. Tremors and ataxia of all limbs present. Scanning speech. Pupils contracted and fixed. Knee- and ankle-jerks absent.

URINARY SYMPTOMS.—Patient states that there is no incontinence, but hospital attendants report continual dribbling of urine. No retention. F.=D.4/N.0.

CYSTOSCOPY.—Urethra almost anæsthetic. Residual urine 480 c.c. Bladder filled with 200 c.c. fluid. Mucosa normal. No cystitis. No marked trabeculation. Funnel-neck bladder (*Type IV*). The colliculus was visible even though the prostatic urethra was not greatly dilated. Bladder distended to 800 c.c. fluid, at which point mild discomfort was felt. The prostatic urethra was still not widely dilated.

Case 15.—T. H. Aged 36. Tabes dorsalis.

Date of primary syphilis unknown. Lightning pains in his legs and arms for two years. Difficulty in walking in the dark. Pupils pin-point, no reaction to light. Knee-jerks very sluggish. No loss of sensation. Hyperæsthesia over arms and back. Wassermann test positive. Has received antisyphilitic treatment.

URINARY SYMPTOMS.—Difficulty in micturition. Believes desire to micturate is subnormal. Strains to urinate, stream poor and interrupted. May pass only a trickle of urine or at other times one or two pints. Following urination bladder does not feel empty. Catheterizes himself. Frequency varies from once a week to twice daily. No dribbling. No nocturnal enuresis, but his bowels have occasionally moved during sleep.

CYSTOSCOPY.—No residual urine. Low urethral sensibility. Feels pull on ligaments. Bladder filled with 200 c.c. fluid. Cystitis, very slight. Trabeculation practically absent. No funnel-neck deformity. Distension increased to 350 c.c., but no change in appearance of bladder neck.

Case 16.—W. J. Aged 60. Tabes dorsalis.

Gonorrhœa thirty years ago. Developed a stricture six years ago and attended a genito-urinary clinic. Tabetic gait noticed at this time and diagnosed as tabes dorsalis. Wassermann test (blood) positive (+ +). Has since received continuous treatment for neurosyphilis. At present marked ataxia of lower limbs. Pupils small, very slight reaction to light. Knee-jerks present.

URINARY SYMPTOMS.—Absence of desire to micturate. Can urinate freely. Never retention. No dribbling. No enuresis. F.=D.3/N.0.

CYSTOSCOPY.—Urethra relatively anæsthetic. Residual urine 30 c.c. Bladder filled with 200 c.c. fluid. No cystitis. Beautiful fine trabeculation all over roof of bladder. Funnel-neck present (*Type IV*). The bladder was further distended, and when it contained 700 c.c. slight discomfort was felt. The dilatation of the prostatic urethra had to some extent increased. Excellent view of verumontanum and prostatic utricle.

Case 17.—J. O. Aged 46. Tabes dorsalis.

Date of primary syphilis unknown. Diagnosis of tabes dorsalis established one year ago. Argyll Robertson pupils. Knee- and ankle-jerks absent. Ataxia. Romberg's sign positive. Wassermann test (blood) positive. For past year has been receiving treatment for neurosyphilis.

URINARY SYMPTOMS.—Normal desire to micturate. Good stream. F.=D.4/N.0. No retention or difficulty. No dribbling. Enuresis on four or five occasions about twelve months ago. No trouble since.

CYSTOSCOPY.—No discomfort on insertion of cystoscope. Residual urine 450 cc. Bladder filled with 200 c.c. fluid. No cystitis. No abnormal degree of trabeculation. Mild degree of funnel-neck (*Type II*). Bladder distended to 500 c.c. without discomfort. Funnel-neck deformity developed considerably, the colliculus now being well seen (*Type IV*).

Case 18.—G. P. Aged 34. G.P.I. (early).

Primary syphilis 1922. Received treatment for one year. Epileptiform seizure six months ago; mentally confused for twenty-four to thirty-six hours, after which his mind made a complete recovery and he had no residual paralysis. The blood Wassermann at this time gave a doubtful positive reaction, the cerebrospinal fluid was strongly positive. He now complains of unsteadiness in the dark. Jaundice developed one month ago and has persisted. The pupils are not abnormally contracted, and react to light and accommodation. Knee- and ankle-jerks present. Sensation normal.

URINARY SYMPTOMS.—No retention. Slight dribbling of urine for two years, not sufficient to soil his clothes. F.=D.4/N.0.

CYSTOSCOPY.—Bladder filled with 200 c.c. fluid. The mucosa was tinged a distinct yellow (jaundice). Very little trabeculation—within normal range. On withdrawing the the cystoscope into the urethra the field appeared dark at first; on further withdrawal the colliculus was well seen in a brightly illuminated cavity (*Type III*). Bladder distended to 600 c.c. without discomfort. Fully developed funnel-neck (*Type IV*).

Case 19.—W. E. W. Age 40. G.P.I.

Primary syphilis twelve years ago. Received three injections at this time but no further

treatment. Two years ago became very nervous and developed marked tremors. Memory also became impaired. Later attended Salford Municipal Clinic, where a diagnosis of G.P.I. was made. Wassermann test (blood) positive. The cerebrospinal fluid contained 106 cells per c.mm.; globulin + + +; Wassermann and Kahn tests positive. Colloidal gold test showed a paretic curve. Has received a year's treatment for cerebrospinal syphilis, including malarial treatment six months ago. Pupils now unequal and give no reaction to light. Knee- and ankle-jerks all present and normal. Sensation appears normal, no loss in saddle area.

URINARY SYMPTOMS.—Normal desire to micturate. No difficulty; no straining. F.=D. hourly/N.0–1. No history of retention. Sexual intercourse normal, normal discharge.

CYSTOSCOPY.—480 c.c. urine withdrawn from bladder (represents two hours). Urine clear. Bladder filled with 200 c.c. fluid. Moderate degree of trabeculation of ordinary type, came and went. No cystitis. Funnel-neck (*Type III*) present. Verumontanum seen. Bladder distended to 400 c.c. without increasing degree of funnel-neck deformity.

Case 20.—V. C. H. S. Aged 50. G.P.I.

Contracted syphilis in Palestine during World War. Four years ago was diagnosed to be suffering from G.P.I. Shortly afterwards was admitted to an asylum suffering from delusions and paralysis of his right side. Wassermann test (blood and cerebrospinal fluid) positive (+ +). Received malarial treatment and made a good recovery. Discharged from asylum and commenced to attend Salford Municipal Clinic for treatment for neurosyphilis. At time of present examination has received two and a half years' treatment. Working for past two years and feels well. The pupils are small, the left reacts sluggishly to light, the right is fixed. The knee- and ankle-jerks are present. No anæsthesia demonstrated over legs or saddle area.

URINARY SYMPTOMS.—Normal desire to micturate. No dribbling. No enuresis. No retention. F.=D.4/N.0.

CYSTOSCOPY.—Urethra almost anæsthetic, no discomfort at cystoscopy. Residual urine 30 c.c. No cystitis present. No trabeculation seen. Bladder neck dilated (*Type II* or *IV*). It was possible to see the urethra extremely well for half to three-quarters of an inch. Though the verumontanum was then visible the surrounding space was inconsiderable. The bladder was distended to 800 c.c., when slight discomfort was felt. No change seen at bladder neck.

Case 21.—C. C. Aged 67. G.P.I.

Primary syphilis eighteen years ago. Attended urological clinic complaining of frequency, and incontinence of urine. No marked psychical changes. Face rather mask-like. Speech slurred. Pupils contracted, no reaction to light. Knee- and ankle-jerks increased. Slight spastic ataxia. No marked loss of sensation. Wassermann and Kahn tests (blood) positive (+ +).

URINARY SYMPTOMS.—Incontinence, clothes soaked with urine, apparently not a continuous dribble but active incontinence. F.=D.6/N. approx. every 20 min. Difficulty in voluntary micturition.

CYSTOSCOPY.—Urethral sensation not completely lost. Residual urine 525 c.c. (urine passed three-quarters of an hour previously). Bladder filled with 200 c.c. fluid. Mild injection of mucosa. Beautiful fine trabeculation all over sides and vault of bladder. Atrophic appearance. Slight degree of prostatic enlargement, small middle lobe present. Funnel-neck urethra (*Type IV*) very marked. Excellent view of colliculus. The degree of funnel-neck deformity was much too great to be accounted for by the minor degree of prostatic enlargement. Bladder distended to 600 c.c. No discomfort. Condition of bladder neck unchanged.

Case 22.—H. A. Aged 45. G.P.I.

No history of venereal disease. Attended hospital complaining of urinary incontinence. During past year has suffered from epileptiform seizures—two to four attacks weekly. Many facial cuts. Face mask-like. No marked psychical changes. Argyll Robertson pupil. Abdominal reflexes very brisk. Knee- and ankle-jerks increased. Extensor plantar response on right side. No definite loss of sensation. Wassermann test (blood) positive (+ +); Kahn test positive (+).

URINARY SYMPTOMS.—Incontinence; slight but continual dribbling of urine for past two years; wets his clothes; more severe at night; increased by active movement. No

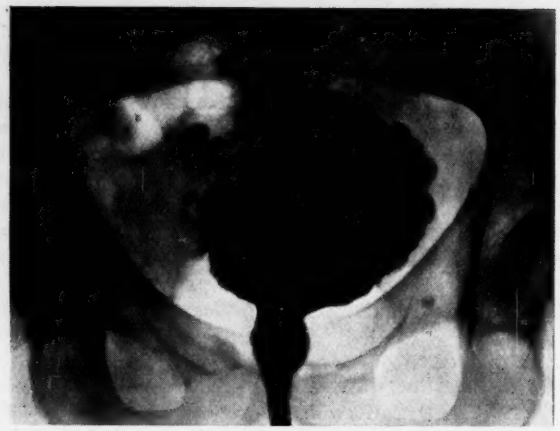

FIG. 364.—*Case* 22. G.P.I. Cystogram showing bladder filled with 200 c.c. potassium iodide solution. Resting pressure 30 c.c. of water. Cystogram made whilst viewing the deformity through the cystoscope.

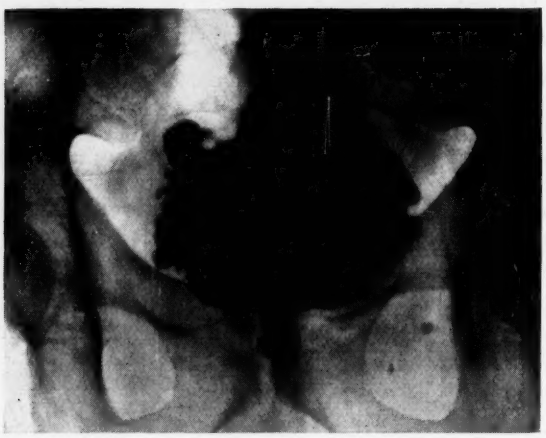

FIG. 365.—*Case* 22. G.P.I. Cystogram showing condition as in previous figure but with cystoscope removed.

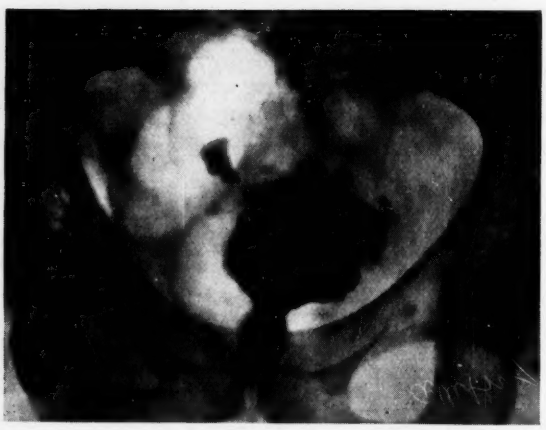

FIG. 366.—*Case* 22. G.P.I. Cystogram following partial emptying of the bladder by voluntary effort, showing the deformity still present.

active incontinence. Voluntary micturition difficult; may wait ten minutes for act to commence; straining; doubles himself up; interrupted stream. F.=D.3/N.1–0. Impotent.

CYSTOSCOPY.—Little diminution in urethral sensation. Residual urine 420 c.c. Urine clear. Bladder filled with 200 c.c. fluid. No cystitis. Well-marked fine and coarse trabeculation, false diverticula. Very pronounced funnel-neck deformity (*Type IV*). Trigone merges almost imperceptibly into posterior urethra. Magnificent view of verumontanum.

FIG. 367.—*Case* 22. G.P.I. Excretion cystogram. There is no sign of a funnel-neck deformity.

CYSTOGRAPHY.—Solution introduced with cystoscope in position (200 c.c.; resting pressure 30 cm. water). Cystogram, taken whilst observing deformity through cystoscope, showed a large funnel-neck deformity (*Fig.* 364). Following removal of cystoscope further cystograms still showed the deformity (*Fig.* 365). An attempt was made to introduce 200 c.c. more fluid through a catheter, but the patient complained of severe irritation in the bladder, and was unable to retain the fluid. A final cystogram with a very small quantity of fluid in the bladder still showed the deformity (*Fig.* 366). A subsequent excretion cystogram, however, showed no sign of funnel-neck (*Fig.* 367).

Case 23.—J. W. G. Aged 37. Cerebrospinal syphilis (Erb's syphilitic spinal paralysis).

Date of primary syphilis unknown. Ten months ago commenced to suffer from spastic weakness in his right foot. A month or two later both legs were involved and marked urinary symptoms were present. Diagnosed as Erb's syphilitic spinal paralysis five months ago. Spastic gait. Pupils give sluggish reaction to light. Knee- and ankle-jerks unusually active. Ankle- and patella-clonus present. Extensor plantar response on stimulation. Some anæsthesia over lower limbs but not well marked. Wassermann and Kahn tests positive (+ +). Received five months' treatment for neurosyphilis prior to examination.

URINARY SYMPTOMS.—Incontinent for past eight months. Urine does not dribble away; suddenly micturates and soaks his clothes (active incontinence). Nocturnal enuresis. Does not feel normal impulse to micturate. No retention. F.=D.3–4/N.1 (and several times into his clothes).

CYSTOSCOPY.—Urethral sensation very low. Residual urine 420 c.c. Bladder filled with 200 c.c. fluid. No cystitis present. Well-marked and very pretty trabeculation of bladder. Funnel-neck (*Type IV*). Colliculus well seen.

Case 24.—W. J. M. Aged 48. Cerebrospinal syphilis.

Gonorrhœa twenty years ago. Attended urological clinic one year ago on account of diurnal and nocturnal incontinence of urine and also marked frequency of micturition. Sudden onset of symptoms. Bladder distended and palpable for approximately 1½ in. above the pubis. Patient also complained of difficulty in walking for the previous fifteen months.

Toes tended to catch the ground. No lightning pains. Pupils normal. Knee-jerks present (+ +). Wassermann reaction positive (+ +).

A cystoscopic examination was performed at this time. The bladder was distended to 200 c.c. The mucosa was hyperæmic and showed marked trabeculation. The posterior urethra was deepened and dilated. The colliculus was enlarged and easily visible (*Type IV*).

The patient has now received antisyphilitic treatment for one year.

CYSTOSCOPY.—Residual urine, 480 c.c. No cystitis. Trabeculation very marked. Funnel-neck (*Type III*). Definite waist intervening between the bladder and the dilated posterior urethra. Colliculus well seen.

II. OBSERVATIONS ON THE NEUROLOGICALLY HEALTHY MALE

This work has been carried out on patients attending the Cystoscopic Clinic for routine examination. All those who were known to be suffering from nervous disease, or who subsequently developed signs of such, have been excluded. Cases of prostatic hypertrophy have also been rejected. The results have been divided into two series (*Table II*). Series I (87 cases) represents a period when no special routine observation for the deformity was kept on the vesical orifice, and the phenomenon was noted in only 3·5 per cent of cases. In Series II (240 cases) a routine examination was made in every patient, and a funnel-neck deformity (*Type III* or *IV*) was found in 7·5 per cent.

Table II.—CYSTOSCOPY IN THE NEUROLOGICALLY HEALTHY MALE.

	SERIES I	SERIES II
Number of males	87	240
Cases of funnel-neck deformity	3	18
Percentage	3·5	7·5

In *Table III* (p. 526) details of those patients exhibiting a positive phenomenon are recorded. Unfortunately, as so frequently happens in clinical investigation, absolutely complete data have not been obtained in each case. Thus when a funnel-neck deformity has been detected under local or sacral anæsthesia, it has not always been possible to confirm its presence on a subsequent cystoscopy.

Weakness of the internal sphincter in the absence of nervous disease requires explanation. Fatigue and straining to micturate are two possible causative factors, but every endeavour has been made to exclude them.

It is our routine practice to use either local anæsthesia (1–1000 percaine) or, more rarely, spinal or sacral anæsthesia, and therefore all patients have had one or other of these anæsthetics. Of the 21 positive cases, 12 exhibited the phenomenon following either spinal or sacral anæsthesia. The number of patients to whom such anæsthetics are given is small, and therefore the large number exhibiting the phenomenon suggests that this form of anæsthesia is of etiological importance. The fact that in three cases where a confirmatory cystoscopy was carried out the sign was absent, also supports this view (*Cases* 4, 13, 17). In *Case* 4 the urethral orifice was rather patulous, but this was probably accounted for by the fact that a small papilloma at the urethral orifice had been destroyed by diathermy, with possible partial destruction of the internal sphincter. In two instances (*Cases* 3 and 9) the deformity was present in the absence of an anæsthetic.

To the remaining 9 cases local anæsthetics were given. The presence of the deformity in the absence of the anæsthetic was confirmed in three instances (*Cases* 11, 18, and 20) (*Fig.* 368). In a fourth case the regurgitation of fluid up the left ureter on a cystogram suggested some lack of tonicity in the bladder wall. In *Case* 11 the Wassermann reaction is positive, and although there are no neurological signs the patient is commencing to suffer from slight incontinence and is probably an early tabetic. These cases suggest that local anæsthesia may cause

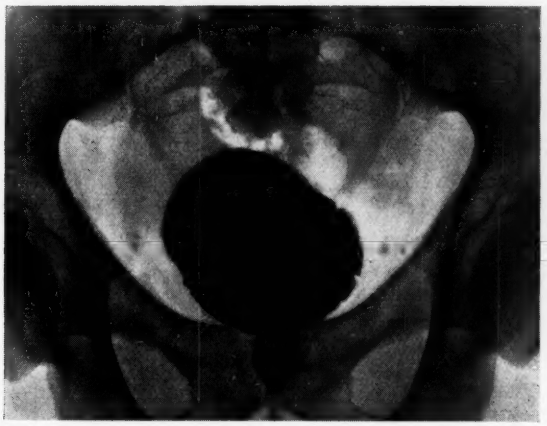

FIG. 368.—*Case* 18. Cystogram from a non-neurological patient showing funnel-neck deformity.

funnel-neck deformity, but that it is of less importance than sacral or spinal anæsthesia in this respect. A pronounced degree when encountered under local anæsthesia points to the advisability of further cystoscopic and neurological examination.

Excluding all doubtful and unconfirmed cases there remain four instances of funnel-neck deformity (*Cases* 3, 9, 18, and 20) in which no cause for the lesion can be demonstrated. It is thus probable that the sign may appear from time to time in the neurologically healthy male, and that it is not definitely pathognomonic of nervous disease.

CONCLUSIONS

1. Funnel-neck deformity of the bladder is very frequently observed in syphilitic disease of the central nervous system. The work of other writers shows that it is not confined to this complaint. The phenomenon is due to paresis of the internal vesical sphincter.

2. Dilatation of the internal vesical sphincter occurs during micturition, and thus minor and transient degrees of the phenomenon may be observed during straining at cystoscopy. Spinal, sacral, or local anæsthesia may produce varying degrees of the condition. The sign is therefore not absolutely pathognomonic of nervous disease, and is only of diagnostic significance when these factors are absent.

3. The deformity may rarely occur in the apparently healthy male.

4. Although visible on cystoscopic examination, cystography may fail to demonstrate the sign. This is due either to sufficient tone remaining in the internal sphincter to close the bladder neck, or to the elasticity of the tissues in this region.

Table III.—NON-NEUROLOGICAL CASES SHOWING FUNNEL-NECK DEFORMITY

No.	Ref.	Age	Disease	W.R.	Anæsthetic	Cystoscopy: Degree of Deformity	Confirmatory Cystoscopy without Anæsthetic	Cystogram or Excretion Urography
1	O. T. ..	32	Frequency and dysuria	Neg.	Sacral and evipan	*Type IV*	Resists any examination unless under general anæsthetic	—
2	W. R. ..	50	Urological examination for hæmaturia	Neg.	Sacral ..	*Type IV*	—	—
3	A. N. ..	17	Dysuria	Neg.	1. Sacral .. 2. Spinal ..	*Type IV*	—	Excretion urogram shows funnel-neck deformity
4	J. W. ..	49	Papilloma of bladder	Neg.	1. Sacral .. 2. Sacral ..	*Type IV* .. .. *Type IV*	Patulous urethral orifice (*Type II*)	—
5	T. B. ..	31	L. hydronephrosis ..	Neg.	1. Local .. 2. Local ..	*Type III* .. .. *Type III* or *IV* ..	No funnel-neck. On straining *Type III* developed	—
6	B. M. ..	50	L. ureteric calculus ..	—	Sacral ..	*Type III*	—	—
7	S. W. ..	56	Chronic interstitial nephritis	—	Sacral ..	*Type III* or *IV* ..	—	Excretion urogram shows no abnormality
8	S. H. ..	52	Testicular pain. Frequency	—	Sacral ..	*Type IV*	—	—
9	J. L. ..	54	Vesico-colic fistula ..	Neg.	1. Local .. 2. Spinal ..	*Type IV* .. .. *Type IV*	Funnel-neck deformity (*Type IV*) present	—
10	G. C. ..	38	L. renal colic ..	—	Sacral ..	*Type IV*	—	Excretion urogram showed no funnel-neck deformity

11	C. D. ..	44	Papilloma of bladder ..	Pos. (++)	1. Local .. 2. Local ..	*Type IV* .. .. *Type IV*	Funnel-neck deformity (*Type IV*)	Cystogram: marked trabeculation; slight funnel-neck deformity
12	H. F. ..	37	Chronic prostatitis ..	—	Sacral ..	*Type IV*	—	—
13	E. B. C.	59	Papilloma of bladder ..	Neg.	1. Local .. 2. Sacral .. 3. Sacral ..	No funnel-neck noted. Verumontanum just visible. *Type IV*	No funnel-neck deformity. On straining *Type IV* developed	—
14	G. A. G.	36	Hæmospermia	Neg.	Local ..	*Type IV*	—	No trabeculation. No funnel-neck
15	W. M. ..	26	Renal investigation for L. lumbar pain	Neg.	Local ..	*Type IV*	—	Slight trabeculation. No other deformity
16	H. W. F.	38	L. pyelonephritis ..	—	Local ..	*Type IV*	—	—
17	N. R. ..	36	Papilloma of bladder ..	—	1. Local .. 2. Sacral ..	No funnel-neck deformity *Type IV*	No funnel-neck deformity	—
18	J. B. ..	35	R. renal calculus ..	Neg.	Local ..	*Type IV*. Increased with straining	Funnel-neck deformity (*Type IV*). Increased a little on straining	Funnel-neck deformity (*Fig.* 368). Slight trabeculation
19	O. P. ..	26	Renal investigation for R. ureteric calculus	—	Local ..	*Type IV*	—	—
20	K. L. ..	12	Nocturnal enuresis ..	—	Local ..	*Type IV*	—	Cystogram: no trabeculation; small funnel-neck
21	F. R. ..	26	Glandular hypospadias. Frequency	Neg.	Local ..	*Type IV*	—	Cystogram: no trabeculation; no funnel-neck; regurgitation up L. ureter

5. Ascending cystography is not a satisfactory method of examining the vesical neck. Excretion cystography offers considerable advantages.

In conclusion, the authors sincerely thank Dr. E. Tytler Burke, Dr. J. Dudgeon Giles, and Dr. W. A. Ramsay for their great help in providing material for this investigation.

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THE ASSOCIATION OF INGUINAL HERNIA WITH TRAUMATIC PERFORATION OF THE INTESTINE*

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The object of this communication is to illustrate the relation which inguinal hernia bears to traumatic perforation of the intestine, as the result of direct injury to the hernia or abdominal wall, or indirectly as the result of excessive muscular effort. The association appears to be definite, but only a few references to it occur in the literature, and these chiefly in German.

CASE REPORTS

Case 1.—An insurance agent, aged 51, was admitted on the morning of June 25, 1921, to the Royal Infirmary, Edinburgh. A bilateral inguinal hernia had been present for ten years. On the previous afternoon, in a race at a picnic, the patient had tripped and had fallen heavily upon the abdomen. Although severely winded, he had suffered no immediate great pain, but half an hour later an increasing colic had developed below the umbilicus. During the night the pain had become unbearable, and had spread upwards into the epigastrium. There had been no vomiting, and at no time had there been symptoms referable to either hernia. The temperature was 100·2°, the pulse 90, and the respirations 20 per minute. The skin was tense over a distended abdomen. Both herniæ were reducible. Hyperæsthesia and marked tenderness were elicited in both iliac fossæ, and a general abdominal rigidity was greatest below the umbilicus. There was dullness in both flanks.

At operation, under general anæsthesia, the peritoneal cavity was opened by a right paramedian incision, and found to contain purulent fluid. The dilated coils of intestine were clothed in fibrinous exudate. On separation of two adherent coils of ileum a perforation was found at the mesenteric border of the bowel. This perforation had apparently occurred initially between the leaves of the mesentery, which had ruptured later to permit general peritoneal infection. A clamp having been applied to the base of the perforated loop, the perforation was closed and invaginated, and the mesentery repaired. The peritoneal cavity was drained through a transverse suprapubic incision. Progress after operation was at first satisfactory, but on the third day hiccup developed, and vomiting, at first of bile and later fæculent fluid, became almost continuous. Death occurred in pyrexia on the fourth day. Post-mortem examination showed a considerable degree of purulent peritonitis, and a general intestinal dilatation.

Case 2.—A greenkeeper on a golf course, aged 71, was carried to the Royal Infirmary immediately after a road accident. Walking home in the roadway under the influence of alcohol, he turned at the sound of a motor-car behind him, and was struck by the car, probably in the belly. He was thrown clear, and rose at once, partly stunned and complaining of severe abdominal pain. There were no striking signs of shock. The pulse was full, strong, and regular, with a rate of 96 per minute. The temperature was 97·4°, the respirations 24 and abdomino-thoracic. The face and nose were brusied. Cerebration was slow and speech incoherent, largely from the effects of alcohol. The abdomen presented no external signs of violence. A reducible right inguinal hernia extended down into the scrotum. The abdomen moved freely with respiration. There was slight tenderness two inches below and to the left of the umbilicus, but no appreciable rigidity. On examination two hours later the abdominal wall was generally rigid, most markedly so to the left of the midline, and tenderness also was generalized.

At operation under general anæsthesia, through a left pararectal sub-umbilical incision, the abdomen was opened and the peritoneal cavity found to contain a small quantity of clear fluid. Tiny subserous hæmatomata were present, scattered over various loops of bowel.

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A loop of small intestine was withdrawn and the gut followed downwards to a perforation, large enough to admit a finger-tip, at the lower end of the ileum. Little or no dissemination of intestinal contents had occurred. Resection of the injured loop was performed, with end-to-end reconstruction. The abdomen was closed and suprapubic drainage of the peritoneal cavity established. Rapid recovery was interrupted by mild infection of the abdominal wound.

Case 3.—A grocer's assistant, aged 52, was admitted to the Royal Infirmary on May 23, 1934. A few hours before admission he had slipped in descending from a tramcar, and had saved himself from falling only by a sudden violent hyperextension of the trunk. He experienced a severe pain simultaneously in the epigastrium and in a right inguinal hernia which had been present, reducible, for years. The pain abated a little, but returned with suddenly renewed severity an hour later, to spread from the epigastrium rapidly over the abdomen, and to persist until admission to hospital. Vomiting occurred once. Increasing indigestion, present for years, and taking the form of epigastric pain an hour after food, had culminated in a recent hæmatemesis, treated in hospital. The temperature was 98°, the pulse-rate 90. The patient, slightly cyanosed, and groaning from pain, lay motionless on his back. Respiration was shallow and entirely thoracic. The abdomen was slightly distended, uniformly and absolutely rigid, and generally tender. Tenderness was maximal in the epigastrium, and over a right inguinal enterocele, even after reduction of the contents of the hernia sac. Both flanks were dull to percussion, and the liver dullness was lost. The case was regarded as a typical one of perforated peptic ulcer. The history of injury was considered coincidental, and the hernial tenderness ascribed to the share taken by the hernia sac in the suspected general peritonitis.

Under general anæsthesia, through a right paramedian incision, the peritoneal cavity was opened and a large quantity of bile-stained fluid evacuated by suction. A fibrinous peritonitis was present. No perforation or ulceration was found in stomach or duodenum. Seven inches below the duodeno-jejunal junction was found a transverse tear of the gut, involving the anti-mesenteric border and two-thirds of the circumference. Through the tear the mucous membrane was pouting, gripped by the torn edges of the muscle layer. The perforation, which had occurred through healthy bowel wall, was closed in two layers, and the wound sutured after passage of a suprapubic drain into the rectovesical pouch. Recovery was uninterrupted.

Case 4 (by permission of Mr. I. H. McClure).—A ship's engineer, aged 56, while returning to his ship one evening, felt sudden severe pain over the symphysis pubis, where the pad of a truss inadequately and only partially controlled a long-standing right inguinal hernia. This pain increased, and rose gradually higher in the abdomen. Vomiting occurred and was repeated. When seen by Mr. McClure four hours later, the patient persisted in blaming his hernia for the attack of pain. It was obvious, however, that an early general peritonitis was present, and laparotomy was performed. A large quantity of turbid fluid was evacuated from the peritoneal cavity. The peritoneum was everywhere congested, and numerous petechial hæmorrhages were present. The lower ileum was considerably dilated, and a circular perforation was found, 4 mm. in diameter, in the antimesenteric border of the bowel a few inches above the ileocæcal valve. The perforation was 'punched-out', and there was no pouting of the mucosa. The perforation was closed and a temporary ileostomy performed above the closure. The patient was discharged three weeks later after an uneventful recovery.

DISCUSSION

Rupture of the intestine as a complication of ill-advised attempts to reduce an incarcerated inguinal hernia, or from direct trauma to the hernia, or even from the pressure of a truss, is not excessively uncommon, and over a hundred cases of general peritonitis from this cause have been reported, chiefly during the last century.[4, 5, 12, 19, 20] Much less common is the variety of rupture of the bowel which, after abdominal violence or sudden muscular strain, may occur as a complication of inguinal hernia. It is this rare injury with which we are specially concerned; our first three cases bring to 33 the total number of reports of its incidence. While it is impossible to estimate the frequency of the condition, it is probably commoner than this figure suggests. Tschistosserdoff[29] observed that in

4 of his 52 cases of ruptured intestine a hernia was present, and Rubritius[21] had 1 instance of the co-incidence in 13 injuries of the intestine. Taylor,[28] in 4 cases of perforation of the intestine, records in 3 the presence of a hernia, but fails to mention its possible importance.

It is interesting to note that the condition has not been observed in women; all the reported cases are in well-built, strong, and healthy men. Traumatic perforation of the intestine has never been recorded in a case of femoral hernia, or in an external hernia other than inguinal. The hernia has usually been old-standing, and usually, but not invariably, right-sided. The perforation may follow direct injury to the abdomen (as in our first two cases, and in most of the reported cases); it may result from sudden violent muscular strain (as in *Case* 3 above).

The symptoms are those of a rapid, spreading, perforative peritonitis; localization by adhesions is rare. Frequently, after the initial pain, there is a latent interval in which the signs and symptoms of peritonitis may be almost entirely absent. The hernia is reducible in the majority of cases, and in all four cases reported above. There may or may not be special tenderness over the hernial swelling.

With the exception of Scarpa's case[23] (in which the colon was injured) the damage has been confined to some part of the small intestine. Although most common in the lower ileum, rupture may occur at any level. In most cases the perforation is situated at or close to the antimesenteric border; in only two instances (Campbell's[4] and the first case above) has it occurred at the mesenteric attachment. The perforation may be either longitudinal or transverse, and the neighbouring bowel may present a bruised appearance, or a split in its serous coat. In most cases, the mucosa pouts slightly outwards, and is gripped by the edges of the opening in the muscle coat.

Except in one case in which the colon was injured within the sac,[4] the perforated loop of intestine has invariably been found within the abdominal cavity. For this reason it is obvious that laparotomy rather than herniotomy is the treatment of choice. Herniotomy is favoured by certain German authors,[7, 21, 27] and its frequent success is due mainly to the proximity, in most cases, of the injured loop to the abdominal inguinal ring. Usually the perforation is sufficiently small to permit closure and invagination by seromuscular suture, but occasionally an almost circumferential rupture demands resection of the damaged loop. In this, as in all traumatic ruptures of the small intestine, the mortality is high—in the neighbourhood of 66 per cent.

The mechanism of rupture of the intestine by direct or indirect violence in cases of inguinal hernia is not always obvious, and it is unlikely that a single explanation can be applied to all cases.

1. Where the causative violence has been applied directly to the hernia (as in our fourth case), the explanation is simple. The bowel is ruptured as it lies unprotected in the hernia sac.

2. It is more difficult to explain why the presence of a hernia should render the intestine more susceptible to injury by a force applied to a point on the abdominal wall remote from the hernia. It can be presumed that a fixed loop of bowel is more liable to injury than is a freely movable loop which can be dislodged in safety by a force applied to the abdomen.[13] The first two cases recorded above immediately suggest that in these a loop of lower ileum, fixed on the one hand at the ileocæcal junction, fixed on the other at the abdominal inguinal ring by a

contraction of the abdominal muscles around the neck of the hernial sac, is drawn relatively taut. It may be broken across by the force applied to the abdomen, or, fixed in this way, it may be crushed between the external force and the posterior abdominal wall. The numerous bruises which presented on the serous surface of the injured bowel loop in our second case are strong proof that in that case, at least, some such mechanism was responsible. In our second case, the site of the tear at the mesenteric border suggests that the point of the abdominal fixation of the affected loop has been its own mesenteric attachment, a possibility which Kahn[11] has previously suggested.

3. Ruptures of the intestine resulting from violent hyperextension of the trunk, without direct injury to the abdomen, are still less easily explained. To account for these cases (which are well exemplified by the third case reported above), Bunge has advanced an ingenious hypothesis. He points out that any sudden rise of intra-abdominal pressure is invariably associated with a coincident rise in intra-intestinal pressure. In the normal abdomen such a rise of intra-intestinal pressure is of little consequence, since the pressure within the bowel is equal to the pressure around it at every point on its surface, and no rupture can occur. If, however, at the time of sudden elevation of intra-abdominal pressure a loop of bowel overlies the mouth of a hernia sac, and a rise of pressure consequently occurs in that bowel loop, the loop lacks the support of the intra-abdominal pressure at a localized point on its surface, protrudes downwards into the sac in which no rise of pressure has occurred, and bursts at that unsupported point. When the intra-abdominal pressure falls again, the ruptured loop moves away from the abdominal ring and discharges its content through the perforation to give a general peritonitis. A neat illustration of this mechanism is afforded by an inflated balloon placed in the barrel of a syringe. Provided the nozzle of the syringe be closed by the finger the balloon will fail to burst, however hard the piston is compressed upon it. As soon as the finger is removed from the nozzle the balloon protrudes through the nozzle where it is unsupported and bursts at that unsupported point. Bunge applied this explanation to all cases of hernia complicated by rupture of the intestine, whether by direct or by muscular violence, but the theory has its greatest value in explaining the rare cases in which the rupture occurs at a time when the hernial protrusion is not present.

Ingenious as the theory is, its complexity is a bar to its unqualified acceptance. The causative muscular violence is almost invariably a violent hyperextension of the trunk. It would be simpler to explain these cases by overstretching of a loop of bowel, fixed at one end at the ileocæcal or duodenojejunal junction, and gripped at the other by contraction of the abdominal muscles around the hernial orifice. In our second case the perforation was seven inches below the ligament of Treitz. Bunge supports his theory by describing such injuries to the intestine as ' ruptures by bursting ', but the direction of a perforation of the bowel and protrusion of the mucosa through a perforation are no certain indication of the mechanism of rupture. There is nothing in the descriptions of reported perforations of the small intestine in cases of hernia to prevent them being considered as ' ruptures by tearing '.

In conclusion, a loop of intestine lying within a hernia sac may be injured by violence applied directly to the hernial swelling—the pressure of an ill-adjusted truss, or too vigorous attempts to relieve an incarceration. Ruptures of the bowel caused by violence to the abdomen appear to be due to the application of the traumatic force to a doubly fixed and immovable bowel loop. Ruptures by

muscular violence are most simply explained by the overstretching of a bowel loop between the ileocæcal (or duodenojejunal) junction and the abdominal inguinal ring, and distraction of these points of fixation from each other by violent hyperextension of the trunk. Bunge's attractive theory of a bowel loop bursting outwards through the neck of a hernia sac (where the support offered by the intra-abdominal pressure is lacking) need be applied only to the excessively rare cases where the hernia sac, though unquestionably present, is empty at the moment of injury.

SUMMARY

In cases of abdominal injury followed by the clinical features of peritonitis, where an inguinal hernia is present, a rupture of the intestine should be suspected.

This rupture may be due to violence applied to the hernia itself, to direct abdominal injury, or, more rarely, to violent hyperextension of the trunk.

The mechanism responsible for the rupture of the bowel varies with the nature of the causative violence.

The first two cases of this series were patients of Mr. J. M. Graham, and the third case was also treated in his wards in the Royal Infirmary, Edinburgh. The author is grateful for permission to publish these cases, for access to their case-reports, and for the advice Mr. Graham has given in the preparation of the paper. The fourth case is published by permission of Mr. Ian H. McClure, of Kirkwall, Orkney, who has been generous also in providing a complete record of it.

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CHRONIC PEPTIC ULCER OF THE ŒSOPHAGUS: A REPORT OF EIGHT CASES

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VARIOUS kinds of ulceration are found in the œsophagus. In this paper I do not propose discussing the malignant, syphilitic, or tuberculous ulcers, or even the so-called decubitus ulcer of the post-cricoid region, but shall confine myself to the simple chronic ulceration found usually in the lower end of the organ and frequently known as chronic peptic ulcer of the œsophagus.

Opinions as to the frequency of this interesting condition differ rather widely. Tileston, [1] in 1906, was able to collect only 8 cases from the literature. Stewart[2] stated that he had found only 1 case in a series of 10,000 post-mortems performed over a period of eighteen years. Stewart and Hurst,[3] in their handbook on peptic ulceration, added 11 more cases to Tileston's 8. Chevalier Jackson,[4] in 1929, by the routine use of the œsophagoscope in all cases of slight dysphagia, found 21 active ulcers and the scars of 67 in a series of 4000 cases. Excellent reproductions of the œsophagoscopic appearances of certain of them are given in his paper. His work has since been criticized by other writers, who have concluded that many of the ulcers described were probably acute in type. Friedenwald, Feldman, and Zinn,[5] in 1929, came to the same conclusions as Jackson about the relative frequency of the condition, and published 13 cases from their own practice in which the condition was diagnosed by œsophagoscopy and X-ray examination. Eusterman, Moersch, and Camp,[6] in 1930, reported 3 cases, the ulcer being situated at the junction of œsophagus and cardia in one, and at the lower end in the other two, causing partial stricture. The condition has also been described by Aurelius[7] in 1931.

Clinical Features.—The three main symptoms are pain, hæmatemesis, and dysphagia.

1. PAIN.—This is referred usually to the lower end of the sternum, sometimes to the epigastrium. It may radiate from the point of origin downwards into the epigastrium, upwards on to the sternum, round to the back, or to the left supra-clavicular region.[8] Barclay,[9] in 1915, by the use of X rays after a barium meal, showed that the pain was caused by spasm of the lower end of the œsophagus, which was due to the passage of food over the ulcerated area. Œsophageal pain appears in many cases to be much worse than even the severe pain of certain gastric and duodenal ulcers; and in a case of œsophageal ulceration reported by Hurst,[10] the patient became extremely neurasthenic because of it. "When a meal was brought into his room, his face became anxious and his hands shook. Several minutes elapsed before he could persuade himself to lift the fork in his trembling hand to his lips; he masticated much longer than was necessary in order to put off the moment of swallowing as long as possible, at the same time shaking his head. He had lost much weight and strength and was extremely depressed."

We should expect the pain in all cases to appear during or immediately after the swallowing of food, especially if it is simply due to spasm of the œsophagus. This is not always so, however, and, as was pointed out by Balfour,[8] it may appear as late as two or three hours after food, so that the lesion may be confused with gastric or duodenal ulcer. He reports a case illustrating this, in which the pain and other symptoms disappeared on treating the ulcer with the application of 20 per cent silver nitrate and adhering to a bland diet, followed by dilatation of the lower end of the œsophagus. In such cases the late pain is probably due to the regurgitation of acid gastric juice during the late stages of digestion.

2. HÆMATEMESIS.—Only very rarely is this a rapidly fatal hæmorrhage, and when so is usually due to erosion of the aorta. More commonly there is intermittent vomiting of ' coffee-ground ' material or frank red blood. Marked anæmia may develop in such cases, and in that of Zahn's[11] the clinical diagnosis made was one of pernicious anæmia.

3. DYSPHAGIA.—In the early stages of the condition this is by no means always present, but when so is due to spasm, the food being felt to stick for a few moments and then pass on. In the late stages of the condition, when fibrous stricture appears, the condition becomes much more severe, and finally complete.

Complications.—The main complications are :—

1. PERFORATION.—As in carcinoma of this region, perforation may occur into the pleura, pericardium, or bronchus. A case reported by Trotter,[12] in 1850, ruptured into the pericardium and caused death in twenty-four hours. Rupture into the left bronchus took place in Finlayson's case.[13]

2. ACUTE MEDIASTINITIS.—This is an uncommon complication of the condition and is due to spread of infection from the base of the ulcer into the surrounding tissues. In Lindsay's case[14] a mediastinal abscess developed and finally ruptured into the lung.

3. DEVELOPMENT OF CARCINOMA.—Ortmann,[15] in 1901, and Tileston[1] have each described the development of carcinoma in the scars of old ulcers of the œsophagus.

Diagnosis.—Apart from the clinical signs and symptoms of the condition, which are only too often far from diagnostic, there are two methods of diagnosis at our disposal.

1. ŒSOPHAGOSCOPY.—By this means the ulcer may be viewed under direct vision, and it was thus that Chevalier Jackson[4] diagnosed his active and healed-ulcer cases.

2. X-RAY EXAMINATION AFTER A BARIUM MEAL.—The crater of a chronic œsophageal ulcer was first visualized thus by Friedenwald, Feldman, and Zinn,[5] in 1929. Jackson showed the presence of spasm in many of his cases, but did not succeed in showing the crater of the ulcer. Hurst,[10] in 1934, demonstrated the ulcer in both of his cases by the barium meal, in one of them only after three former X-ray examinations had failed to show any lesion. In one case the cardiac sphincter was seen to be widely open, immediately above was a large shadow representing the crater of the ulcer, and, still higher, constant spasm of the œsophagus was apparent. In the second case he stresses the importance of the enormous gas bubble seen in the cardiac end of the stomach, which was due to œsophageal spasm interfering with the escape upwards of swallowed air in the same way as it interfered with the passage downwards of ingested food.

CASE REPORTS

In view of the comparative rarity of the condition, we feel that the following 8 cases of chronic simple ulceration of the œsophagus are worth recording. They have been met with in the course of 1500 post-mortems done at the Glasgow Royal Infirmary over the past four years. Cases of acute ulceration and ante-mortem digestion have, of course, been excluded; and all the cases recorded showed evidence of chronicity in the form of fibrous induration extending outwards from the ulcer and in many cases endarteritis of vessels.

Case 1.—Mrs. O'H., aged 75 years, was admitted to hospital on Jan. 14, 1936, with signs of acute obstruction of the bowels. She was operated upon as an urgent case and a cæcostomy performed. Cellulitis of the surrounding abdominal wall developed, and death took place from sepsis on Feb. 13, 1936. The patient stated in her history that she had had periodic attacks of indigestion for many years, but these had never been severe. There was no dysphagia.

POST-MORTEM REPORT.—The body was that of a woman of medium build and rather poorly nourished. A collection of pus was present between the layers of the abdominal wall in the region of the operation wound. The abdominal cavity showed only some adhesions binding the terminal ileum to the brim of the pelvis. On the anterior and posterior walls of the stomach, close to the middle of the lesser curvature, were chronic ulcers. That on the anterior wall had muscle in its base and showed marked fibrous induration around. That on the posterior wall was slightly less advanced. Each measured 1·5 cm. in diameter.

The œsophagus was opened from behind. It was normal in calibre, but at its lower end was an area of chronic ulceration extending completely round the tube. It measured 2·5 cm. in length and its lower edge coincided with the cardiac sphincter except at its left side, where it extended as a longitudinal ulcerated area measuring 2 by 1 cm. into the cardiac end of the stomach. The upper edge of the œsophageal ulcer was very poorly defined and faded off irregularly. The lower edge was more sharply seen. In the base of the ulcer the circular muscle of the œsophagus was apparent as transverse markings. Some thickening of the tissues outside the œsophagus was apparent. Besides the chronic ulcer there was also present a late ante-mortem digestion of the œsophagus showing some evidence of attempts at healing. The whole of the lower two-thirds of the tube was completely denuded of epithelium, there being simply the submucosa remaining stained light-brown with changed blood. Between this and the upper normal third of the tube was an area containing small rounded islets of epithelium which were obviously growing in an attempt to cover the denuded area.

Histological examination of the ulcerated area showed the appearances of chronic ulceration with some irregular fibrosis extending into the muscular coat of the œsophagus. The upper part of the œsophagus showed ante-mortem digestion, and at parts a thin growing edge of epithelium was seen showing attempts at healing.

The post-mortem diagnosis was cellulitis of the abdominal wall following cæcostomy; chronic ulceration of the œsophagus; ante-mortem digestion of the œsophagus.

Case 2.—Mrs. C., aged 51 years, was admitted to hospital on May 25, 1935, complaining of distension of the abdomen and marked breathlessness. Examination showed a very marked ascites, and multiple tumour masses were felt in the abdomen. Paracentesis was frequently performed, but the patient slowly went downhill and death took place on Nov. 17, 1935. There was no history of indigestion, and while in hospital the patient was fed on light diet with no particular symptoms to suggest an œsophageal lesion.

POST-MORTEM REPORT.—The body was that of a rather spare woman. The left pleural cavity contained 500 c.c. of slightly blood-stained fluid; the right contained 600 c.c. of thin purulent material with a few flakes of fibrin. Both lungs were normal. The peritoneal cavity contained 9 litres of blood-stained fluid. The peritoneal surfaces were studded with nodules of tumour tissue which had spread from a carcinoma of the uterus. The stomach and intestines were normal.

The œsophagus was opened from behind. At its lower end was an ulcer extending almost completely round it. It started a fraction of a centimetre above the cardiac sphincter and extended upwards for a length of 5 cm. On the right posterior aspect, about the middle of

the ulcerated patch, was a roughly circular area of much deeper ulceration, which extended completely through the muscular coats. At its upper edge the ulcer ceased in an irregular circinate manner, so that close observation was necessary to make out the line of demarcation. At its lower end it ceased in a very distinct, almost straight, line. Some induration of the tissues around was felt. On separating the ulcer from the adjacent mediastinal tissues, the base gave way at its deep excavated part. No direct communication was found between the ulcer and the right pleural cavity, but the septic condition of the tissues in the region of the deepest part of the ulcer suggested that the inflammatory process had spread from here through the mediastinal tissues to the right pleural cavity.

Microscopical examination of the ulcer showed involvement of the circular muscle only at most parts. Some increase of fibrous tissue was seen in the base, which contained congested vessels at places. At the deeper area of ulceration the process extended through both muscle coats and into the peri-œsophageal tissues. Much polymorphonuclear infiltration was present in the neighbourhood of this area, which gave the impression of being a more acute ulcerative process superimposed on a chronic ulcer. There was no evidence of malignant disease in the neighbourhood of the ulcer.

The post-mortem diagnosis was carcinomatosis of the peritoneum; chronic ulceration of the œsophagus; acute pleurisy.

Case 3.—Mrs. B., aged 55 years, was admitted to hospital on Feb. 5, 1933, with a hæmatemesis. For four years the patient had been troubled with periodic attacks of indigestion. Each attack lasted for a period of from a few days to three or four weeks, and were separated by relatively free periods. The pain appeared immediately after the taking of food and was referred through to the back, being felt between the shoulder blades. Flatulence was troublesome during the attacks. No vomiting had occurred until a week before her admission to hospital, when her appetite disappeared and she commenced to vomit greenish-mucoid material. High epigastric pain was present at this time and became much worse when she attempted to take food. On the morning of the day of admission she felt very faint and drank 2 drachms of brandy. She immediately felt very sick and vomited some dark blood-stained material. A short time later she vomited some fresh blood and was admitted to hospital. After admission she vomited about 5 oz. of clotted blood mixed with mucus. Examination showed nothing of note apart from slight tenderness in the epigastrium. In 1919 the patient had had oöphorectomy performed, and in 1929 had had an abscess in the lower abdomen drained. Her teeth had all been removed some time previously.

Three days after admission the patient developed a pneumonic consolidation of the right lung. Blood-culture showed the presence of Type II pneumococcus. Death took place on Feb. 18, 1933.

POST-MORTEM REPORT.—The body was that of an elderly, well-nourished woman. An old midline scar was present in the lower abdomen. The cardiac muscle showed cloudy swelling. On the posterior cusp of the mitral valve were some recent vegetations of the ulcerative type. The upper lobe of the right lung was in the condition of grey hepatization. The stomach showed a certain degree of hour-glass contraction which was due to a transverse scar 5 cm. in length lying about the middle of the lesser curvature. No active ulceration was present.

The œsophagus was opened from behind. At its lower end was a chronic ulcer which extended completely round the tube and measured 5 cm. in length. Its lower edge was fairly well defined and corresponded to the cardiac sphincter. The upper edge was irregular and faded off into intact mucosa. Circular muscle showing areas of cicatrization was seen in the base. No bleeding vessel was found. The œsophagus above was of normal size.

Histologically the ulcer showed chronicity in the form of fibrous induration, extending for some distance outwards. Some endarteritis of vessels was apparent. Section of the gastric scar showed a line of well-formed old fibrous tissue.

The post-mortem diagnosis was chronic ulceration of the œsophagus; hæmatemesis; lobar pneumonia.

Case 4.—Andrew J., aged 71 years, was admitted on April 16, 1936, complaining of pain in the abdomen of two months' duration. The pain was situated in the epigastric region and was of an aching character. It tended to appear especially in the evening and was relieved by alkaline powder. The pain had no definite relationship to food, but the patient's appetite had been very much poorer than normal since its onset. There was no vomiting at any time during the illness up to the time of admission. The bowels were somewhat irregular, and on

one occasion a week or two before, the patient noted that the stool had a very black colour. Since the beginning of his illness the patient had lost over 2 st. in weight. His past health had always been very satisfactory and he did not remember having ever been previously confined to bed. Upon admission the patient showed evidence of having recently lost a good deal of weight. His jaws were edentulous and his tongue dry and furred. No epigastric tenderness was present. A blood examination showed the presence of a marked secondary anæmia, the cell-count being red blood-corpuscles 2,220,000, white blood-corpuscles 5,400, the Hb 32 per cent, and the colour index 0·72. After admission the patient periodically vomited small quantities of brown blood-stained material. On April 29, 1936, he vomited 500 c.c. of altered blood and soon afterwards collapsed and died.

POST-MORTEM REPORT.—The body was that of a male subject with a fairly large frame but showing much emaciation. The skin was very pale. The heart showed slight hypertrophy of the left ventricle. Both lungs showed marked œdema posteriorly. The prostate showed marked enlargement, due to adenomatous hyperplasia, and the urinary bladder showed hypertrophy and dilatation. The stomach contained 300 c.c. of dark-red blood-clot. No ulceration was present in the stomach or duodenum and the blood had obviously come from the œsophageal ulcer.

FIG. 369.—*Case* 4. Diffuse chronic ulceration of the œsophagus, which is very deep in one area and has eroded the aorta.

In the lower 8 cm. of the œsophagus was an area of ulceration extending completely round the organ. At its upper end the ulcer extended upwards as two rounded areas with a tongue-shaped process of mucous membrane running downwards for a short distance between them. On its right side the ulceration was fairly superficial, and circular muscle could be seen in its base at places. On this side the ulcer faded off above into intact mucosa, the exact line of demarcation being very indefinite. On its left side the ulcer was much deeper over an area measuring 5 cm. in length and 2·5 cm. in breadth. The edges of this deeper area of ulceration were very steep in their inferior parts, but showed some terracing in the upper part. Near the lower edge of this part was an oval, very deeply ulcerated patch measuring 0·8 by 0·3 cm. which had as its base the intima of the aorta; and on holding the specimen up to the light a series of small cracks could be seen through which oozing of blood had taken place (*Fig.* 369).

Histological examination showed the appearances of chronic ulceration with much fibrous induration.

The post-mortem diagnosis was chronic ulcer of the œsophagus eroding the aorta.

Case 5.—Bernard McA., aged 53 years, was admitted to hospital on Dec. 29, 1932, suffering from hæmatemesis of four days' duration. For thirty years the patient had suffered from indigestion. The attacks lasted for weeks or months at a time and were separated by free intervals, which had lately been becoming shorter. Epigastric pain appeared from an hour to an hour and a half after the ingestion of food and lasted for a variable period, being frequently relieved by the taking of more food. Vomiting at first had been infrequent, but had been becoming more frequent during the preceding four years. Upon admission the patient was found to be an emaciated man who looked older than his years. He continued for some days to vomit 'coffee-ground' material and to pass changed blood per rectum. As he was going downhill rapidly it was decided to cauterize the bleeding ulcer. On Jan. 11, 1933, a large blood transfusion was given and, thereafter, the abdomen opened under local anæsthesia. "Kissing' ulcers were found on the anterior and posterior walls of the first part of the duodenum. That on the posterior wall was bleeding and was cauterized. A posterior gastro-enterostomy was then performed. Death took place a few hours after operation.

POST-MORTEM REPORT.—The body was that of an emaciated, elderly looking man. On the anterior and posterior aspects of the first part of the duodenum were chronic ulcers measuring 1·5 cm. in diameter. The floor of the posterior one was formed by pancreatic tissue. Slight pyloric stenosis was present and the stomach was a little hypertrophied and dilated. At its lower end the œsophagus was adherent to the tissues outside. It was opened from behind. In its lower end towards the right side was an area of chronic ulceration. This was roughly circular, measuring 3·5 by 4 cm., with edges which were rather serpiginous except the lower one, which was clean cut and ran in an almost straight line transversely across the lower end of the œsophagus. At its right side this edge lay just above the cardiac sphincter, but towards its left sloped slightly upwards from it. Growing from this edge was a small sprouting mass of what looked like granulation material. Compared with this lower edge the upper and lateral ones were indistinct and faded off into intact mucosa. The ulceration was comparatively superficial in its upper part and circular muscle was exposed in its base. In the central and lower parts, however, the process had penetrated much deeper, cicatricial

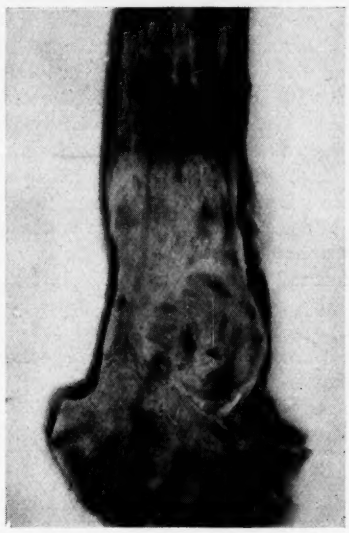

FIG. 370.—*Case* 5. Chronic ulcer of the œsophagus. Note the sharply defined lower edge where the ulcer is deepest. Circular muscle is exposed elsewhere.

FIG. 371.—*Case* 6. Chronic ulcer of œsophagus. Circular muscle and overgrown fibrous tissue are seen in the base.

fibrous tissue lying in the base. The œsophagus above the ulcer appeared slightly dilated and hypertrophied, but showed no other abnormality. (*Fig.* 370.)

Histological examination of the ulcer showed the appearances of chronic ulceration, there being much fibrous tissue formation with small collections of round cells. Endarteritis of vessels was seen near the surface.

The post-mortem diagnosis was chronic duodenal ulcers; hæmatemesis; chronic ulcer of the œsophagus.

Case 6.—John K., aged 69 years, was admitted on Feb. 7, 1935, complaining of pain in the upper abdomen and vomiting of six weeks' duration. For many years the patient had been troubled with indigestion, which was marked by pain coming on at a variable period after food, flatulence, and water-brash. Six weeks before admission his condition became very much more urgent. The pain and especially the vomiting became very severe, the former having no definite relationship to food, and becoming, if anything, worse after it was taken. The patient had had pneumonia in 1916 and acute cholecystitis in 1933. He was a moderate smoker and used to drink a large quantity of alcohol. This had, however, ceased some months before admission. The abdomen was slightly scaphoid and the skin dry and loose, suggesting fairly recent loss of weight. The tongue was moist with a slight white fur. All the teeth

had previously been extracted except two which appeared healthy. Slight tenderness was present over the whole abdomen. There was some anæmia present of the secondary type, the red blood-cells being 3,030,000 and the white cells 5800 per c.c. The Hb was 60 per cent. A gastric analysis showed complete absence of hydrochloric acid, with a total acidity slightly above normal. X-ray examination after a barium meal showed a moderate amount of dilatation of the stomach, but no evidence of ulceration. During the patient's residence in hospital melæna was almost constantly present. He improved slightly under treatment, but on Feb. 28, 1935, he developed signs of internal hæmorrhage from which he died on the same day.

POST-MORTEM REPORT.—The body was that of a poorly nourished looking man. The stomach was very greatly dilated, with hypertrophied wall. Immediately beyond the pylorus was a deep chronic ulcer on the posterior wall of the duodenum 2 cm. in diameter and with over-hanging steep edges. The base was formed by pancreatic tissue, and the ends of several small arteries were visible. Much fibrosis extended from the ulcer into the pancreas and lesser omentum. Black altered blood was present in the ileum and a small quantity was also found in the colon. The sigmoid loop showed long-standing diverticulitis. The gall-bladder contained twelve faceted gall-stones of the mixed type.

The œsophagus was opened from behind. Its mucous membrane was very pale in appearance. There was an area of ulceration involving the lower 5 cm. of the organ and running completely round it. The lower edge of the area lay just above the cardiac sphincter and was fairly well defined. The upper edge faded off in a series of ill-defined arcades. In this superficial ulceration were three longitudinal areas of much deeper excavation, each measuring about 1·5 cm. in breadth and showing cross striation in its base due to exposure of circular muscle fibres. At their lower edges, i.e., near the cardiac sphincter, these areas were specially deep but became much shallower above. It was the upper edges of these areas which caused the arcade-like appearance already mentioned. The wall of the œsophagus in the region of the ulceration was much thickened, fibrosed, and adherent to the structures around, so that separation was effected only with difficulty. Above the ulcer the œsophagus showed marked hypertrophy and dilatation. (*Fig.* 371.)

Histological examination of the ulcer showed it to have the characters of a chronic ulcer involving the circular muscle coat at most places, which showed varying degrees of fibrosis with collections of round-cells here and there.

The post-mortem diagnosis was chronic ulcer of duodenum with hæmorrhage; chronic œsophageal ulcer.

Case 7.—John M., aged 71 years, was first admitted to hospital on Aug. 18, 1926, complaining of loss of appetite, sickness, and vomiting of six weeks' duration. Except for occasional slight attacks of indigestion the patient had had good health previous to this time. For the first few weeks of his illness the vomiting had occurred about one and a half hours after food, but had gradually been getting much worse, and just before admission was occurring ten minutes after each meal. It was present even when he was on light diet. Abdominal pain was absent except for a slight feeling of discomfort in the upper abdomen. He had lost 1½ st. in weight during the six weeks of his illness. The patient admitted that he had previously taken a fairly large quantity of alcohol every week. He smoked 3 oz. of tobacco per week. Upon admission the patient was found to be a rather thin elderly man. There was evidence of recent loss of weight. A neoplasm of the stomach was suspected, but X-ray examination of the gastro-intestinal tract was negative. He was put on light diet, his sickness ceased, and he commenced to put on weight. He was discharged on Sept. 16, 1926, feeling very well and with no return of symptoms.

On Sept. 1, 1931, the man was readmitted to hospital. He stated that shortly after his discharge five years before his stomach had recommenced to trouble him. This took the form of periodic attacks of epigastric pain and vomiting, the pain appearing at a variable period after food, often coming on soon after its ingestion. He found that heavy foods such as potatoes and meat aggravated his symptoms and he had been avoiding these. For the five days preceding admission the symptoms had become very urgent, the pain in the epigastrium being very severe, and hiccup was almost constantly present. The day before admission he vomited a quantity of 'coffee-ground' material. Examination showed the patient to be a rather spare, elderly man. Constant hiccup was present. The patient had no teeth. The breath was foul and the tongue thickly coated with a dirty, brownish fur. Deep pressure in the epigastrium elicited some tenderness but no mass was palpable. The lungs and urinary

and nervous systems were normal. The patient appeared a little confused and gave only a very indefinite account of the symptoms. He improved under very light diet, and on Sept. 8 the sickness had ceased and the hiccup was only occasionally present. He was being given 3 pints of milk and a ½ pint of Benger's food daily. On admission the stools contained obvious melæna, but on Sept. 12 it was only found on careful chemical examination. Examination of vomited material on Sept. 13 showed free hydrochloric acid (titrated with N/10 NaOH) 50; total acidity, 78. On Oct. 19, 1931, the improvement had persisted and the patient's mental condition was much clearer. He complained that his food stuck near the lower end of his sternum after he had swallowed it. An X-ray examination after a barium meal was carried out and the report was "there is obstruction of the œsophagus, the appearance being highly suggestive of neoplasm". An œsophagoscopic examination was carried out on Dec. 2, 1931. No carcinoma of the œsophagus or fundus of stomach was seen. The walls of the œsophagus appeared rather closer together than normal, suggesting an extrinsic pressure on it, but no decisive finding was obtained. The patient was now put on 5 min. of tincture of belladonna three times daily.

FIG. 372.—*Case* 7. Diffuse ulceration of œsophagus, the wall being greatly thickened by fibrosis. The lower edge is fairly well defined, but the ulceration fades off in the upper part. Probes are in two vessels which had been bleeding.

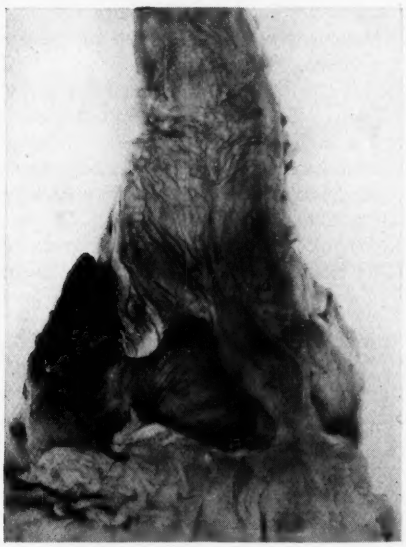

FIG. 373.—*Case* 8. Peptic ulcer of œsophagus. Note the deep overhanging edge and the tongue-shaped process of heterotopic gastric mucosa extending upwards between the lateral edges of the ulcer. The base is formed by the pleural surface of the left lung.

This improved his œsophageal condition and he was able to swallow his food normally after this. On Dec. 7, 1931, he was discharged from hospital feeling very well.

On Aug. 30, 1932, the patient was admitted to hospital for the third time. He complained of hæmatemesis and epigastric pain of one day's duration. He said that he had been keeping fairly well since his discharge from hospital less than a year previously. Four days before his present admission he developed epigastric pain accompanied by marked nausea. Alkaline treatment only helped him slightly. On the day preceding admission the pain was very much more severe and the patient vomited brownish material which contained blood. Examination showed him to be a moderately well nourished elderly man. His complexion was florid and his mucous membrane of good colour. He complained of vague high abdominal pain but no rigidity was present. Soon after his admission he had a copious motion which contained

altered blood. While in hospital he had various attacks of vomiting, the vomitus consisting of partly digested food. On Sept. 11 he had two attacks of hæmatemesis which weakened him considerably. He also complained of pain high up in the epigastrium shooting through to the back. On Sept. 27 it was noted that he continued to vomit about once daily and that the vomited material always contained some altered blood. This vomiting appeared to be unaffected by any medicinal treatment. His general condition became poorer, he became confused mentally, and died on Nov. 5, 1932.

POST-MORTEM REPORT.—The body was that of an adult man of rather poor nutrition. The heart and lungs showed no abnormality apart from senile degenerative changes. The stomach showed slight dilatation, but no ulceration, recent or old, was present. No other abnormality was found in the abdomen.

Externally the œsophagus felt greatly thickened in its lower half and was firmly adherent to the tissues outside, so that it could not be completely separated from them. It was opened from behind. The lower two-thirds of the organ showed very marked thickening of its wall due to replacement by firm fibrous tissue, and measured 7 mm. in thickness at parts. The mucous membrane was completely absent in the lower half. The ulcerated area ceased abruptly in a straight line at the cardiac sphincter, but at its upper part it faded gradually off into intact mucosa, there being a 'transitional' area where islets of epithelium started to appear, showing some attempt at healing. The base of the ulcer consisted mainly of cicatricial fibrous tissue, but at parts the cross striation of the circular muscle coat was apparent. Two small perforations into vessels were found in the middle of the ulcerated patch. Blood exuded from the lower one on slight pressure. Just outside the œsophagus, on the left side 3 cm. above the cardiac sphincter, was an enlarged gland the size of a bean containing much anthracotic pigment. (*Fig.* 372.)

Microscopical examination of a piece of tissue taken from the middle of the ulcer was carried out. The remains of longitudinal muscle were present at one end of the section. It showed a well-marked increase of fibrous tissue between its fibres. The fibrous tissue was dense and hyaline at parts but became more cellular and of more recent origin nearer the surface. Many of the vessels showed marked endarteritis obliterans. There was no evidence of malignant or syphilitic change. In the fibrous tissue deep to the muscular coats numerous lymphoid follicles with prominent germ centres were seen.

The post-mortem diagnosis was chronic ulcer of the œsophagus with hæmorrhage.

Case 8.—Patrick C., aged 58 years, was admitted on June 8, 1935. For years his stomach had troubled him, there being an indefinite history of pain after food accompanied by occasional vomiting. An X-ray photograph was taken, the suspected diagnosis being peptic ulcer; but no abnormality of the stomach or duodenum was seen. For some months before admission the patient's disability had become extreme. He was afraid to take food because of the pain, had become very neurasthenic, and had taken to alcohol as a means of relieving the pain. There was the occasional vomiting of 'coffee-ground' material. Anæmia had become very marked, and he was admitted to hospital with a condition of spasm of the vessels of the fingers resembling Raynaud's disease. Upon admission the patient was very weak and breathless. He admitted having drunk half a bottle of whisky an hour or so before admission. He died fairly suddenly four hours later.

POST-MORTEM REPORT.—The body was that of a middle-aged male in the last stages of emaciation. In the posterior aspect of the lowest lobe of the right lung was an area of red hepatization. A less advanced degree of pneumonia was also present in the middle lobe. All the organs of the body showed evidence of inanition. The marrow of the femur showed a well-marked erythroblastic reaction.

Marked thickening was present in the tissues around the lower end of the œsophagus, so that carcinoma was suspected on external examination. The organ was opened from behind. At its lower end, 1 cm. above the cardiac sphincter, was a deep oval ulcer, measuring 5·5 by 4 cm., almost completely encircling the circumference of the organ and leaving only a small patch of intact mucosa 1·5 to 2 cm. in breadth. The edges of the ulcer were very steep and overhanging at parts. The base was firm and infiltrated, and at its left side was composed of the pleural surface of the left lung, which showed some fibrosis at this part. Gentle manipulation broke down the adhesions binding the ulcer to the pleura, causing a direct perforation into the left pleural cavity, showing how the patient had been living on the verge of a precipice for some time. Closer examination of the mucous membrane in the region of the ulcer

showed the presence of a remarkable state of affairs. The intact mucosa separating the lateral edges of the ulcer was found to be heterotopic gastric mucosa which extended as a tongue-shaped process of well-preserved tissue upwards from that of the fundus of the stomach. (*Fig.* 373.) The two important features were the close resemblance of the ulcer to the typical chronic ulcer of the pyloric region of stomach or first part of duodenum and the presence of the heterotopic mucosa.

Microscopical examination showed marked fibrosis spreading from the base of the ulcer, with complete destruction of the muscle coats in the neighbourhood. On the surface of the ulcer was a thin layer of necrotic material infiltrated with polymorphs and fibrin. Deep to this was a fairly cellular granulation tissue with many fibroblasts. Beneath, this fibrous tissue became more dense and avascular. The heterotopic gastric mucosa bore a resemblance to that found normally towards the pyloric end of the stomach, the glands being fairly short and wide. Oxyntic cells were present, but were comparatively few in number.

The post-mortem diagnosis was chronic peptic ulcer of the œsophagus; advanced inanition; lobar pneumonia.

Table I shows the main features of the 8 cases described in detail above.

Table I.—SHOWING MAIN FEATURES OF 8 CASES OF CHRONIC PEPTIC ULCER OF THE ŒSOPHAGUS

SEX	AGE	SYMPTOMS	ŒSOPHAGUS	GASTRIC OR DUODENAL ULCERATION	CAUSE OF DEATH
Female	75	Periodic attacks of indigestion	Chronic ulcer with ante-mortem digestion	Chronic gastric ulcers	Cellulitis of abdominal wall after cæcostomy
Female	51	No history of indigestion	Chronic ulcer more acute at one part	None	Carcinomatosis of peritoneum, with terminal mediastinitis and empyema due to œsophageal ulceration
Female	55	Indigestion for 4 years: hæmatemesis on admission	Chronic ulcer	Scar of old ulcer across lesser curvature	Lobar pneumonia following hæmatemesis from œsophageal ulcer
Male	71	Indigestion for 2 months	Chronic ulcer of rounded type	None	Hæmorrhage from eroded aorta
Male	53	Indigestion for 30 years	Chronic ulcer of rounded type	Chronic ulcers of duodenum	Death following operation on duodenal ulcers
Male	69	Indigestion for many years	Chronic ulcer	Chronic ulcer of duodenum	Death due to hæmorrhage from duodenal ulcer
Male	76	At first like gastric condition. Later dysphagia	Very chronic ulceration with marked fibrosis	None	Death from constant bleeding from ulcer
Male	58	Indigestion for many years, much pain latterly. Very neurasthenic and alcoholic because of pain	Typical peptic ulcer of œsophagus with heterotopic gastric mucosa	None	Pneumonia following advanced inanition from hæmorrhage and starvation due to ulcer

DISCUSSION

Certain important features emerge in a study of the eight cases which we have just described.

a. Age Incidence.—All the patients were over 50 years of age, the average age being 63·5 years. It is to be noted, however, that the ulcers had been unsuspected during life and some had probably been present for years, so that the true age incidence will be actually lower than this post-mortem figure.

b. Sex.—Five of our patients were males and three were females, so that the sex incidence appears to be slightly higher on the male side.

c. Coincident Ulceration.—In four of the cases ulceration was also present in the stomach or duodenum, the patient having apparently the so-called 'ulcer diathesis'.

d. Symptoms.—In two of the cases the patients had no notable dyspeptic symptoms. In the others, indigestion had been present for a variable period of time, usually for years. In two of these the symptoms were most likely due to the duodenal ulceration which was also present. In one the gastric ulcer had been healed for a long period of time, leaving an avascular fibrous scar, so that the late symptoms were almost certainly due to the œsophageal ulcer. In the remaining three cases the severe dyspeptic symptoms were undoubtedly due to the œsophageal ulceration. It is worth noting that in all of them the symptoms were referred to the stomach and duodenum, not only by the patient, but by the physician; and in only one of them, and that at a late stage when fibrous stricture was taking place, was the œsophagus suspected as the cause of the trouble. In four of our cases hæmatemesis was present, but in only two of them was this a marked feature.

e. Cause of Death.—In five of the cases the œsophageal ulceration could be said to be at least an important factor in causing death. In two of these the actual cause was lobar pneumonia, but in both the ulcer was undoubtedly the exciting factor, in the one case being superimposed on the very advanced inanition which it had produced in the patient, and in the other following a hæmatemesis from the ulcer. In one case the immediate cause of death was empyema and mediastinitis which had spread from the base of the ulcer. In this case chronic ulceration had been present for some time and a more acute process had become superadded, most likely as the result of the extremely debilitated condition of the patient following the malignant cachexia. In another case death was the result of recurring bleedings from the ulcer. In the last case death was due to erosion of the thoracic aorta by the ulcer.

f. Position of the Ulcer.—In all the cases the ulcer was situated at the lower end of the œsophagus, and in many of them its lower edge was sharply limited by the cardiac sphincter.

g. Appearance of the Ulcer.—There appears to be two different types of ulcer found in this region and we shall later suggest two different etiologies :—

1. In the first type the ulceration is fairly superficial in type. It may remain thus as in *Cases* 1, 3, 6, and 7, and in the last case the extreme amount of fibrous thickening of the wall which may ensue should be noted. On the other hand, it may become much deeper at one area, an ulcer within an ulcer as it were, and, as in *Case* 2, cause mediastinitis and empyema, or, as in *Case* 4, erode the aorta. This type of ulcer involves the area of œsophagus immediately above the cardiac sphincter.

The lower edge is in most cases clean cut and unhealing, whereas the upper one is indefinite, often circinate, and fades off into intact mucous membrane.

2. In the second type the ulcer is localized, is very deep and penetrating, and in fact has the typical appearance of a very chronic gastric or duodenal ulcer. The presence of gastric heterotopia in the region of the ulcer is to be noted, and, clinically, the extremely urgent symptoms causing profound neurasthenia and inanition.

h. Diagnosis.—Repeated and very careful œsophagoscopic and X-ray examinations appear to be necessary to demonstrate the condition, and in one of our cases narrowing of the œsophagus was the only definite lesion diagnosed by the œsophagoscope, although at the autopsy a short time later a very advanced ulcerated and fibrous condition of the tube was seen. We have already discussed the X-ray diagnosis of the lesion by visualizing the crater after a barium meal. From the study of our cases we feel that this will be the exception rather than the rule, and that the most certain diagnostic feature is likely to be spasm in the region of the ulcer, for in our series of cases very few had craters large enough to fill with barium.

ETIOLOGY

The two types of ulceration which we have described are so different in their appearances that we are tempted to look for two different causes.

1. The shallow, more diffuse type of ulceration conforms in its position, widespread character, and abrupt cessation at the cardiac sphincter to the acute form of ulceration of the œsophagus called ' ante-mortem digestion '. We have examined specially the œsophaguses from a fairly large number of cases and have concluded that, whereas the acute fulminating type of ante-mortem digestion is fairly rare, the slighter degrees are not uncommon, and may very likely be frequently recovered from, in some cases completely, in others, as we shall suggest, leaving an area of chronic ulceration.

Like burns, ante-mortem digestion can be divided into degrees of severity, examples of all of which have been seen post mortem. The first stage is one of congestion of the œsophagus with no destruction of the mucous membrane. In the second, the mucous membrane has disappeared at places, leaving, however, intact islets of epithelium between. In the third degree the mucous membrane has disappeared completely over a large area of gullet, almost always its lower third. In the fourth or last degree the whole thickness of the wall is necrotic and perforation may take place into the pleural sac. Death in such cases is, of course, invariable. In the second degree of the condition there are islets of epithelium left corresponding to the interpapillary processes of skin in a third-degree burn ; and should the patient recover from his primary illness, these will act as centres from which epithelium will grow to cover completely the denuded area. Such healing cases are sometimes seen post mortem. To the naked eye the islets appear rounded off at their edges, resembling thin patches of white paint on the raw surface, which has by this time lost the dark sanguineous appearance of the acute stage of the condition (*Fig.* 374). Microscopically the epithelium is seen growing as a thin layer over the denuded patch, mitotic figures being seen in the stratum mucosum near the growing edge.

Consider the third degree of ante-mortem digestion—the stage where there is

complete destruction of the mucous membrane of the lower end of the œsophagus, corresponding to a fourth-degree burn. If such a case recovers from the primary illness what will happen to the affected part? The necrotic material will separate and the mucous membrane at the edge of the area commence to grow in to cover the denuded area, but there are no epithelial buds left to fill in the central part, and if we carry our analogy with a burn of the skin still further, we can see how difficult it would be for such an area to be completely epithelialized—how healing usually takes place so far and then ceases until skin-grafts are applied. Still more difficult will it be for healing to take place in the case of the œsophagus, whose mucous membrane is being constantly irritated by food, and on occasions also by regurgitated acid gastric juice. Thus, a stage will be reached when the acute ulcer is replaced by a chronic ulcer of the diffuse and fairly shallow type like many we have described. The upper edge which is farthest from the irritating gastric juice will show more healing than the lower edge which is in close proximity to the gastric contents; the lower edge will be sharp and clean cut, showing little healing as compared to the ill-defined healing upper one.

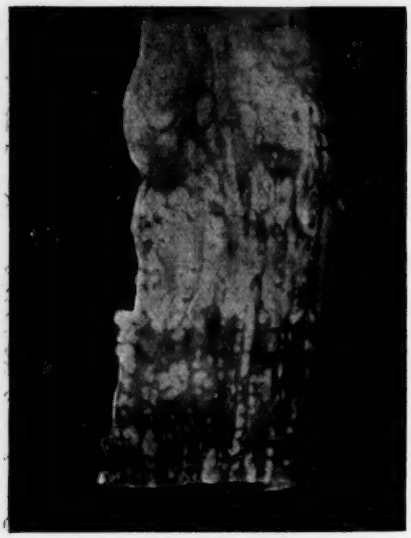

FIG. 374.—Healing ante-mortem digestion of the second degree, showing the rounded edges of the growing islets of epithelium.

As an additional proof of this etiology, we would point out the association of four of our cases with the 'hyperchlorhydric' conditions of gastric and duodenal ulceration; and also that in one of our cases there was, along with the chronic ulceration, a recent ante-mortem digestion of the œsophagus above the chronic lesion.

2. Our second type of ulceration differs very markedly from the first and is shown in its typical form in *Case* 8. It resembles the chronic ulcer found in the pyloric end of the stomach or the first part of the duodenum, and is marked by depth rather than diffuseness. The symptoms also appear to be more urgent and serious in such cases. A clue as to its origin was given by the finding of a large patch of heterotopic gastric mucosa beside the ulcer. Heterotopic mucosa was present in Stewart and Hartfall's case[3] and in Fraenkel's[16] and Tileston's[1] cases. When the patches are small and the amount of acid secreted correspondingly little, it will be rapidly diluted by saliva and cause no harm. Thus heterotopic mucosa is not infrequently found in the œsophagus with no coincident ulceration. If the patches are more extensive, and if there is some degree of spasm of the cardiac sphincter superimposed, the accumulation of this acid secretion in the lower part of the œsophagus will set up an irritative, and later an ulcerative, condition. That is the view usually held as to the association of peptic ulcer of the œsophagus with gastric heterotopia. If that be the correct explanation, we should expect the ulceration to be rather of the diffuse type which we have already described instead of the localized crateriform type. Thus, while not denying that such is the correct explanation, especially where the

heterotopic mucosa occurs in the upper part and the ulcer in the lower part, we also suggest from the appearances of our own case that the ulcer may actually start in the heterotopic mucosa itself, which, as is well known in the case of Meckel's diverticulum, shows a marked tendency to ulcerate when it finds itself in a foreign environment.

SUMMARY

1. Eight cases of chronic ulceration of the lower end of the œsophagus are described.

2. They are discussed and tabulated to show their main features.

3. Two main types are distinguished and the etiology of each of these types is discussed.

My thanks are due to Professors Harrington and Burton and Drs. Middleton, Muir Crawford, and Henderson for the use of the clinical notes of their cases, and above all to Professor J. Shaw Dunn for his very kind and willing help and advice at all times.

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RENAL RICKETS

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DURING the last quarter of a century it has become possible, largely as a result of improved X-ray technique and an extended use of biochemical analysis, to clarify many formerly obscure abnormalities in bone development, particularly perhaps that group designated somewhat loosely as late or adolescent rickets. It is probable that a certain number of these cases were merely examples of hunger osteomalacia, especially those occurring in some areas after periods of famine, the most important recent examples of which were supplied by the post-war period in Central Europe. There remains a group of cases in which rachitic changes appear during later childhood or adolescence, or which persist from infancy into this period in spite of treatment, and which, taken as a group, are not particularly rare, presenting from time to time a diagnostic problem to every alert clinician. This group necessarily includes a few cases of ordinary infantile or vitamin D deficiency rickets in which the nutritional deprivation has remained uncorrected or has made its appearance for some reason at an abnormally late period in the child's life. It also includes two forms of so-called rickets in which certain entirely new factors are concerned, namely, cœliac and renal rickets. With the latter must be considered certain forms of primary hyperparathyroidism in which secondary renal changes occur. Cœliac rickets has been admirably dealt with by Parsons,[1] and will not be further considered here beyond remarking that it is essentially a low-calcium form of rickets due to long-continued loss of vital elements in the stools.

The problem of the pathogenesis of renal rickets remains incompletely solved, and has been complicated during recent years by the discovery in certain cases of enlarged parathyroids. The issue has thus become entangled in the far-flung net of the endocrinologist, who would like to range the disease alongside that type of hyperparathyroidism to which reference has already been made. In view of the rarity of the condition, and of the uncertainty which still surrounds the question of its etiology, it is important that every case in which the diagnosis can be established should be reported as fully as possible.

CASE REPORT

HISTORY.—The patient, a boy of 14, was admitted to Southmead Hospital on April 20, 1936, exhibiting most of the typical features of renal rickets. At birth he was stated to have weighed only 4 lb. He was always regarded as delicate, and remained stunted in growth, but exhibited no deformities until well past the age of seven. He had long suffered from thirst and polyuria, first noticed between two and three years of age. He had had measles at two, and at the age of seven contracted nasal diphtheria, for which he was treated at a fever hospital for thirteen weeks.

Deformities of the legs and feet were first noticed when he was nearly 8, and had become progressively worse, particularly during the last year. The deformities prevented

him from running or playing games, but he had ridden a bicycle right up to the day of admission, and was in other ways physically active. He had never been able to talk properly, and had always been extremely deaf. Notwithstanding all this, his parents, who were very fond of him, insisted that he was as mentally alert as any of their five other children had been at a comparable age period. His diet had always been adequate and varied, except that he disliked and rarely touched meat. The stools were normal.

The mother, aged forty-six, gave an interesting obstetrical history which may be recorded as follows :—

Date of pregnancy	1910	1912	1914	1916	1918	1921	1922	1923	1923
Sex of offspring	♂	♂	♀	♀	♀	M	♂	M	M
Age of offspring	26	24	22	20	18	—	Patient aged 14	—	—

(M = miscarriage)

The child's parents both appeared to be in perfect health, and there were five other living children, none of whom exhibited the slightest deformity or under-development. The Wassermann reactions and Kahn tests on both father and mother were negative, while their serum calcium and phosphate estimations were normal.

On Examination.—The boy weighed 4 st. 7 lb. and was 4 ft. 1 in. in height, figures which are roughly equivalent to those of a boy of eight or nine. He was of a sallow muddy complexion with freckling of the forehead and round the nose. The hair was coarse and dry and somewhat sparse. There was a moderate anæmia of the mucous membranes. The malar bones were unduly prominent, giving a depressed appearance to the bridge of the nose, which was rather squat and broad. The eyes appeared rather sunken by comparison, and were in addition somewhat heavily lidded, so that he had a perpetually sleepy appearance. Another striking feature about the face was the prominence of the superior maxillæ, so that the lower jaw was definitely receding (*Fig.* 375). (It is possible that this peculiar facial conformation represents a type of deformity occasionally occurring in renal rickets, as a somewhat similar appearance has been referred to in a case described by Ellis and Evans.[2])

The whole appearance was suggestive of considerable mental defect, but his intelligence was difficult to assess owing to the deafness and a rather gross speech defect of the lalling type. He was certainly able to lip-read quite well, and his ability to express himself by a wealth of sign and gesture hinted at an underlying mental activity utterly belied by his very unprepossessing exterior.

Both testes, though small, had descended into the scrotum, but secondary sex characteristics were rather infantile. Nothing abnormal was detected in the cardiovascular system. Blood-pressure : 120/75. No definite thickening of peripheral vessels ; eye grounds normal. The kidneys were not palpable.

Deformities.—The most striking abnormality was due to a flexion of both hips, with marked compensatory lordosis of the lumbar spine (*Fig.* 375). This caused a rather waddling gait reminiscent of bilateral congenital dislocation of the hips. Even with the lordosis the effect of the flexed hips was never fully overcome, so that he was not able to stand upright properly. At the ankles there was an outward bending of the lower ends of tibia and fibula on both sides, with a resulting valgoid displacement of the feet (*Fig.* 376). There was moderate genu valgum and a well-marked rickety rosary, while a swollen metaphysis could be palpated at the ends of most of the long bones.

X-ray Examination.—Radiographs of the whole skeleton were taken, and revealed characteristic changes of renal rickets. There was a generalized osteoporosis associated with florid rachitic changes at the extremities, the wide-cupped metaphyses exhibiting that peculiar fluffy or woolly appearance which is so constantly seen in severe cases, and which, as Brailsford[3] has rather aptly suggested, looks as if the cancellous bone has undergone a mucinous degeneration. Many of the bones were bent and contorted, particularly the upper end of the femur and the lower ends of the tibia and fibula. The necks of both femurs showed what appeared to be an almost complete separation from the upper epiphyses (*Fig.* 377). while at the lower ends of the tibia and fibula on both sides the outward rotation of the bones had caused a partial sheering off at the softened epiphysial junction, producing the

extreme valgoid deformity to which reference has already been made (*Fig.* 378). Gross changes of the woolly type were present at the lower end of the radius and ulna, with subperiosteal erosion extending along the shafts (*Fig.* 379). The skull showed a remarkable degree of thickening and rarefaction with considerable stippling and 'honeycombing' (*Fig.* 380).

FIG. 375.—Lateral view of patient, showing facies and extreme lordosis.

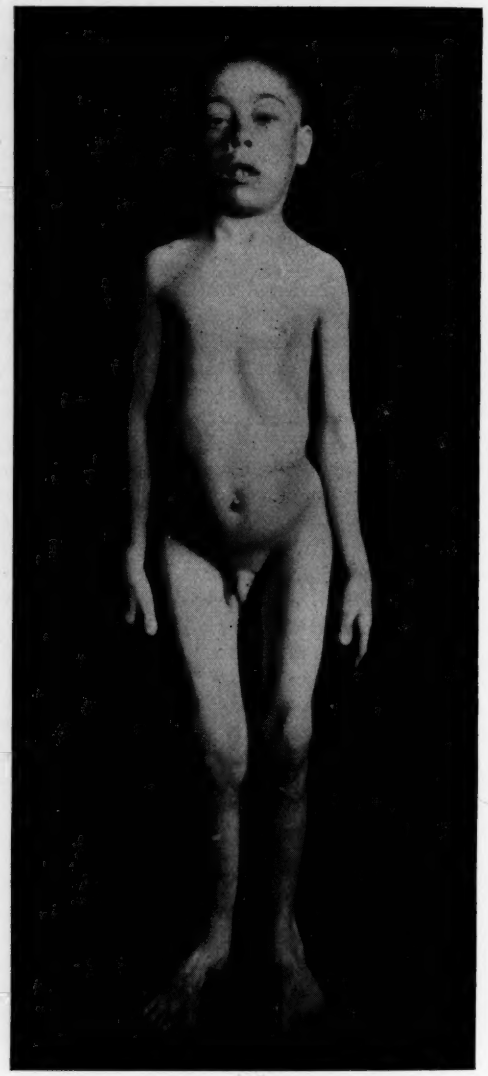

FIG. 376.—Front view of patient, showing facies and valgoid deformity of feet.

RENAL INVESTIGATIONS.—The urine was pale and copious, of uniformly low specific gravity (1000–1004), and contained a trace of albumin and an occasional hyalogranular cast. Urea-concentration tests showed literally no concentrating power, viz., before urea, 0·85 per cent; one hour after, 0·85 per cent; two hours after, 0·8 per cent; three hours after, 0·85 per cent. Water excretion test: after 1 pint, 13 oz. excreted in 4 hours. Maximum specific gravity 1004, minimum 1000.

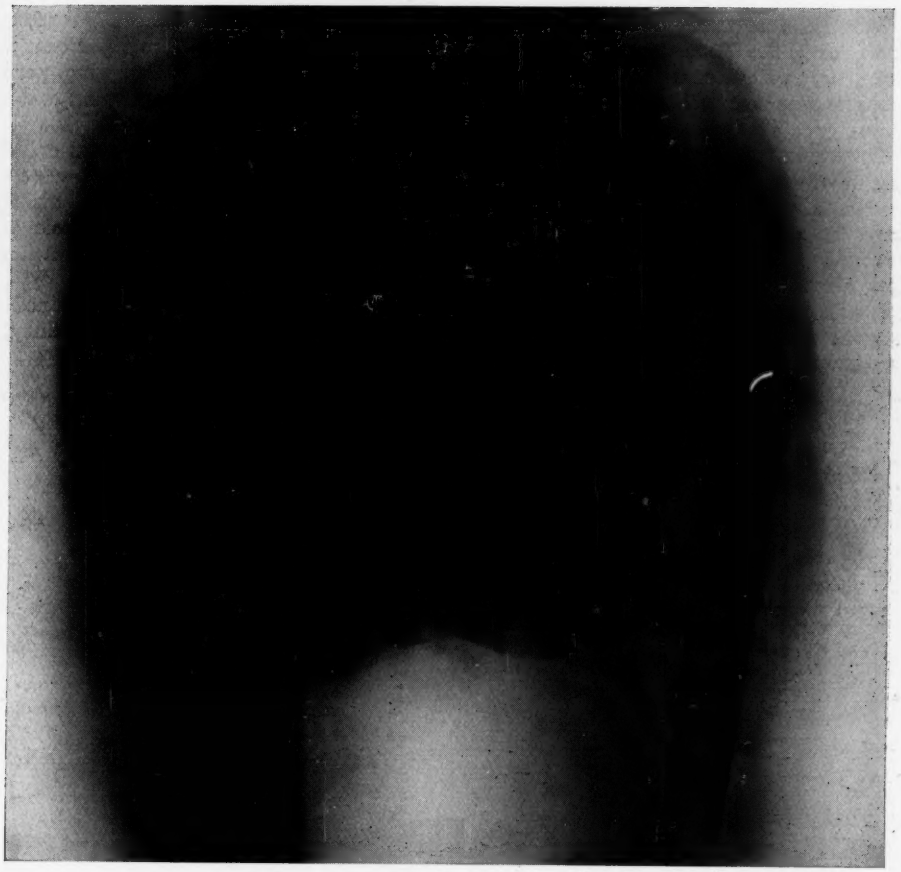

FIG. 377.—Radiograph of hip-joints showing rachitic changes and false appearance of epiphysial displacement.

FIG. 378.—Radiograph of lower end of right tibia and fibula showing collapse of metaphysis and rotation and partial separation of epiphysis.

FIG. 379.—Radiograph of left wrist showing gross rachitic change of the woolly type, with subperiosteal erosion.

BLOOD CHEMISTRY.—Blood-urea 318 mg. on April 29, 1936. On June 15, 1936, a day prior to death, the blood-urea had risen to 397 mg. Serum calcium 13·6 mg. per cent; one month later, 12·5 per cent. Serum phosphates 6·5 per cent; one month later, 5 mg. per cent. Phosphatases 59·6 units (April 29, 1936); 51·1 units (June 15, 1936). Wassermann and Kahn reactions gave a negative result.

COURSE OF THE DISEASE.—In spite of the startling degree of renal inadequacy as evidenced by the blood-urea and renal function tests the boy to all intents and purposes appeared to be in comparatively good health, was bright and cheerful, and frequently amused his fellow patients by a versatile display of pantomime with his hands. The uræmic finale which supervened came with quite unexpected suddenness, beginning with drowsiness and twitching, passing on to convulsions and coma, and terminating fatally in twenty-four hours.

POST-MORTEM EXAMINATION.—Permission for a limited post-mortem was obtained only with great difficulty, so that our examinations were confined to one femur, one kidney, and the parathyroids.

FIG. 380.—Radiographic appearance of skull showing thickening, rarefaction, and 'honey-combing'.

The Femur.—There was a striking disparity between the apparent radiographic appearance (*Fig.* 377) and the finding at post-mortem (*Figs.* 381, 382), for whereas the radiograph suggested a slipped epiphysis at the hip-joint the actual lesion was a collapse of the metaphysis of the neck. In this situation the ordinary cortical and cancellous bone is replaced by a mixture of fibrous tissue and cartilage, in which some bony spicules can be made out. The lines of ossification at both ends of the bone are greatly thickened and irregular, and the hyaline cartilage of the epiphysial line of the head is continuous with the mass of cartilage and fibrous tissue forming the collapsed neck of the bone. The epiphysial line of the great trochanter is also greatly increased in depth, extending deeply into the metaphysis and also joining up with the large mass of fibro-cartilage already mentioned. The lower end of the femur presents no gross deformity, though the epiphysial line is thickened and the bony cortex here thinned and deficient. The thinning of the bony cortex is seen most pronouncedly in the head, where the somewhat thickened articular cartilage appears to have no underlying layer of bone but to be attached directly to the trabeculæ and spicules of the cancellous portion. In the shaft the cortex is spongy and soft, but the marrow cavity appears normal in extent and not unduly vascular.

Microscopically the femoral head shows thinning of the cortical layer of bone (*Fig.* 383). In places calcification and osseous structure is present only as a thin encrustation on the deep aspect of the cartilage, while over large areas it is completely absent. The microphotograph illustrates well the evidences of simultaneous bone formation and absorption as represented

by areas where osteoblasts in rows are apparently concerned with formation of freshly calcified cartilage, while in close proximity to these are bony laminæ showing the occasional lacunæ in which large osteoclasts are producing the typical clear-cut absorption indentations. The

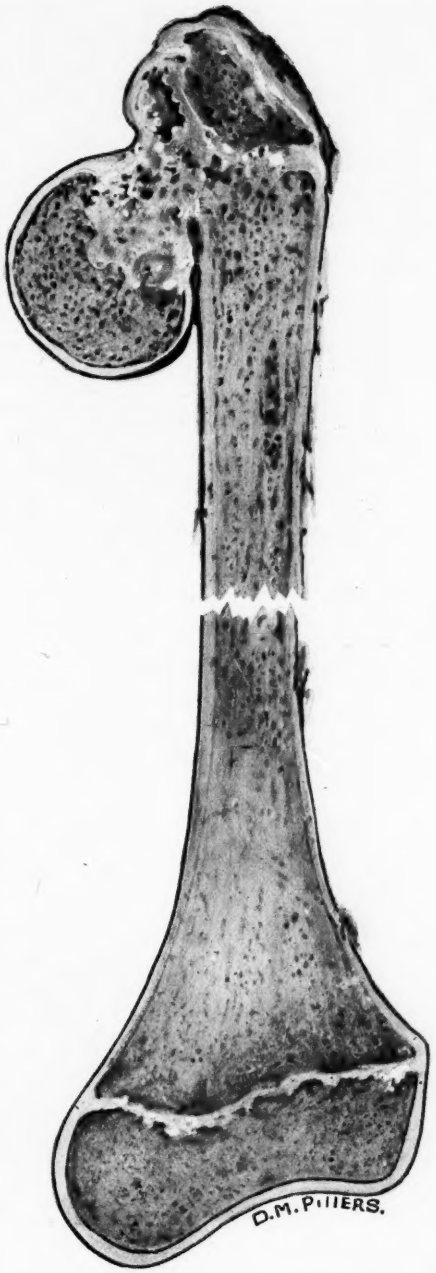

FIG. 381.—Femur, showing the metaphysial collapse of the neck and widened lines of ossification.

trabecular bony spicules show similar evidences of bone formation and absorption, without any signs of osteoid tissue, and the inter-trabecular spaces are occupied by a loose,

well-vascularized, fibro-fatty tissue which becomes more cellular and firm at the periphery. There is no evidence of any hæmopoiesis.

Sections of the epiphysial cartilage of the head (*Fig.* 384) show, on the metaphysial side

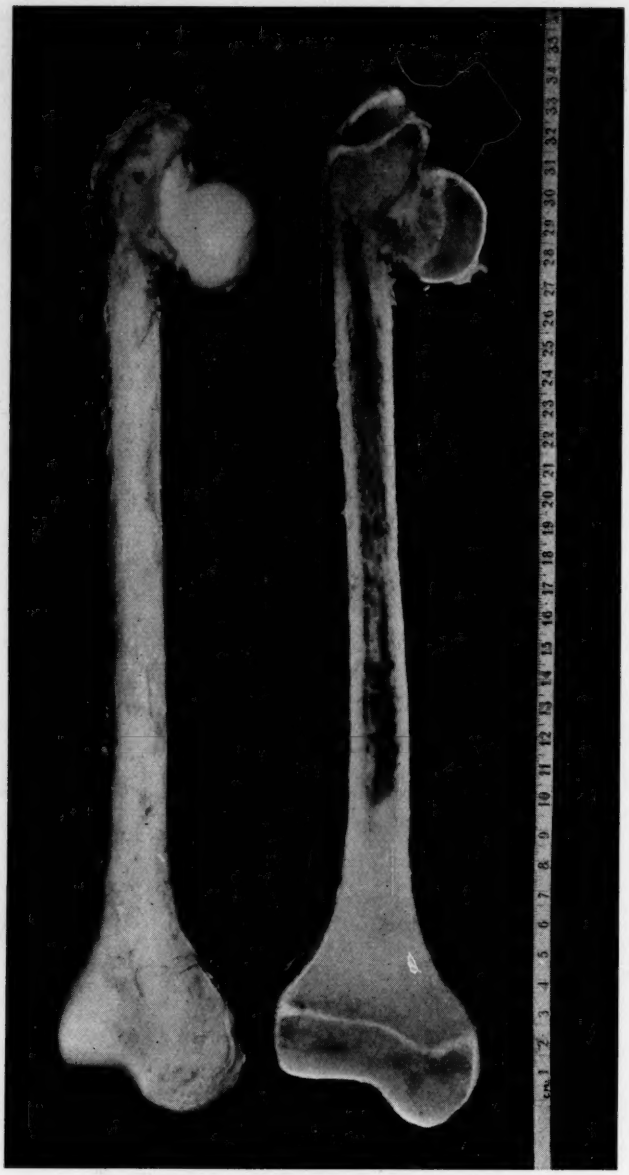

FIG. 382.—Whole femur after longitudinal section.

of the greatly thickened band of hyaline cartilage, an absence of the normal columns of hyperplastic cartilage cells merging into osteoid and bony spicules. Here, as in the cortex of the bone, evidences of absorption predominate. The bony spicules are firm and well formed, and there is practically no osteoid structure. The inter-trabecular matrix is more fibrous than normal.

FIG. 383.—Microphotograph of cortex of head of femur showing the articular cartilage and the scantiness of the cortical layer of bone. (× 50.)

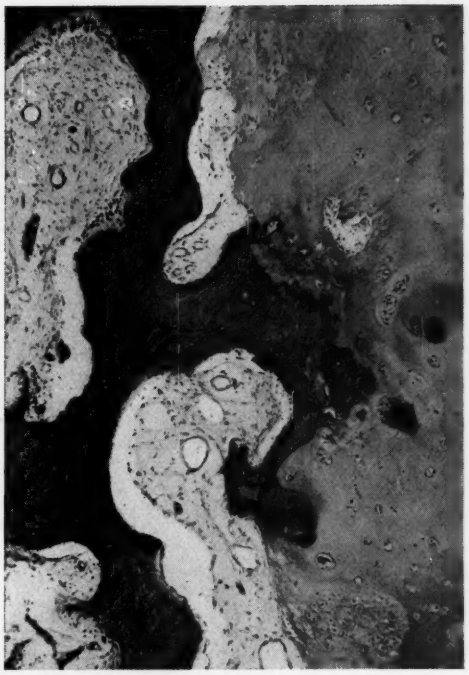

FIG. 384.—Microphotograph of the diaphysial aspect of the epiphysial line of the head of the femur, showing the complete lack of the usual proliferating columns of cartilage cells. (× 50.)

FIG. 385.—Microphotograph of fibrotic tissue constituting the collapsed metaphysis of the neck of the femur. (× 50.)

The microscopical structure of the softened and collapsed metaphysis of the neck is seen in *Fig.* 385. Bony spicules are few, and merge directly, without the intervention of any layers of osteoid, into a fairly dense fibrous tissue in which are numerous large vascular channels. Such activity as is evinced in connection with the bony trabeculæ is almost entirely that of absorption by osteoclasts.

To summarize: The changes in the bone are essentially those of cessation of growth with pronounced bony absorption and demineralization, which have led to weakening at all points of strain, and hence to the broadened splayed epiphyses, and more particularly to the extensive metaphysial collapse, with attempts at compensation by overgrowth of fibro-cartilage from the epiphysial line and articular surfaces. The changes resemble rickets only superficially, and present none of the characteristic microscopical structures associated with avitaminosis D.

The Parathyroids.—As seen in the coloured plate (*Fig.* 386) all four parathyroid glands were enlarged to many times their normal size, their actual measurements being 10 × 6 × 4 mm. and 15 × 6 × 4 mm. for the upper pair, and 15 × 8 × 6 mm. and 11 × 10 × 5 mm.

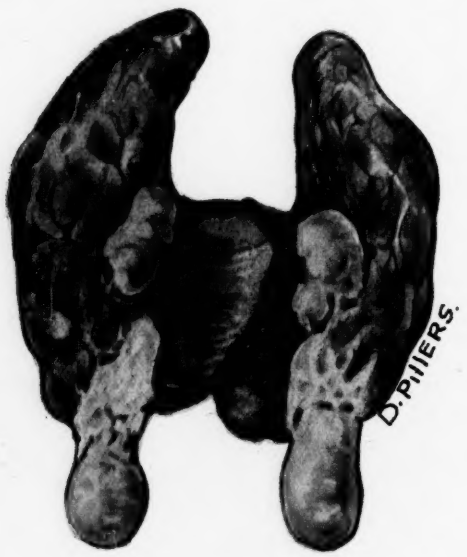

FIG. 386.—Posterior view of the thyroid gland to show the postion and size of the four hyperplastic parathyroids.

for the lower (the average normal size is about 5 × 3 × 2 mm.). In each case the glands presented a homogeneous appearance, and they were regarded as hyperplasias and not as adenomas.

Their microscopical structure presented no abnormal features. The cells were mainly of the principal type arranged in the usual columns and cords, with, here and there, definite alveolar arrangement with appropriate elongation of the cells. The oxyphil cells were not numerous, but also not unusually sparse considering the age of the patient. These oxyphil cells usually occurred in small clumps of two to five, but in a few places formed much larger congeries. One unusual feature presented itself in the section of one of the lower glands, in that the pericapsular tissue consisted of thymus gland. This probably represents an ectopic position of the thymus, though in cases in which the thymus is enlarged it is not unusual to find it extending to the base of the thyroid gland. By reason of the limited nature of the post-mortem examination in this case the size of the thymus was not observed, and the finding of thymic tissue in this situation was quite unexpected. The Hassall corpuscles in this fragment of the gland were very large, and most of them were partially or completely calcified.

The Kidneys.—The one kidney which was obtained for examination was greatly reduced in size and pale in colour (*Fig.* 387). The capsule was slightly adherent, but the surface was

diffusely and evenly granular (*Fig.* 388), the pitting and excrescences being coarser than is seen in chronic nephritis, and resembling closely the irregularity of advanced renal changes of essential hypertension. The cut surface also was pale. The cortex was narrow, the

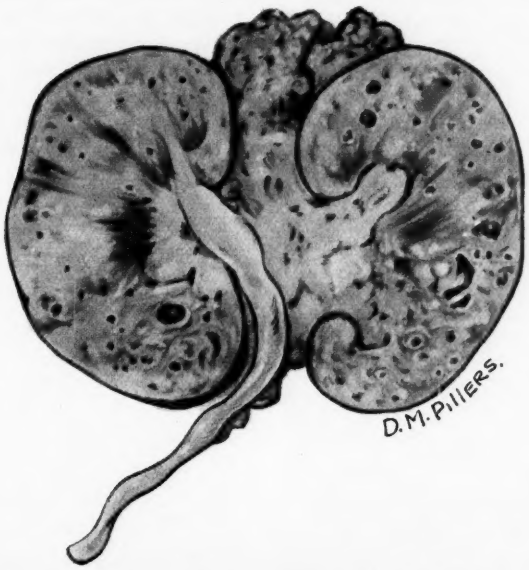

FIG. 387.—Kidney: cut surfaces showing the generalized fibrosis and loss of demarcation between cortex and medulla.

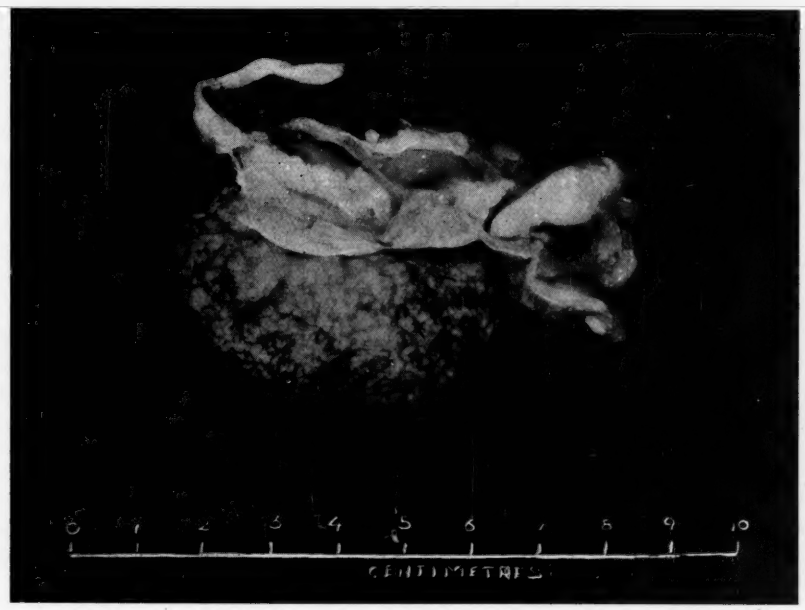

FIG. 388.—Kidney, showing gross reduction in size and the granular pitted surface.

demarcation between cortex and medulla largely lost, and there was considerable increase of pelvic fat. There was no evidence of pelvic or ureteric dilatation, and no pyelitis. The arterial branches and twigs on the cut surface showed no obvious thickening, though the renal artery itself was firmer and more rigid than normal.

Microscopical sections of the kidney present an unusual picture. The general effect (*Fig.* 389) is that of advanced renal sclerosis. Numerous glomeruli are completely obliterated and represented merely by hyaline pink-staining whorls, others are obviously hypertrophied, while still others show varying degrees of atrophic changes and are enclosed in fibrosed thickened laminated Bowman's capsules. The tubules are reduced in number, but greatly dilated, and a moderate number of small cysts are present in the cortex. The tubular epithelium is somewhat flattened. The interstitial tissue is greatly increased in amount, and is widely infiltrated by the usual cells of chronic inflammation, which in places form dense accumulations. The arteries do not show medial fibrosis or hyalinization, nor is there any intimal proliferation. The picture is thus typical neither of chronic nephritis nor of hypertensive nephrosclerosis.

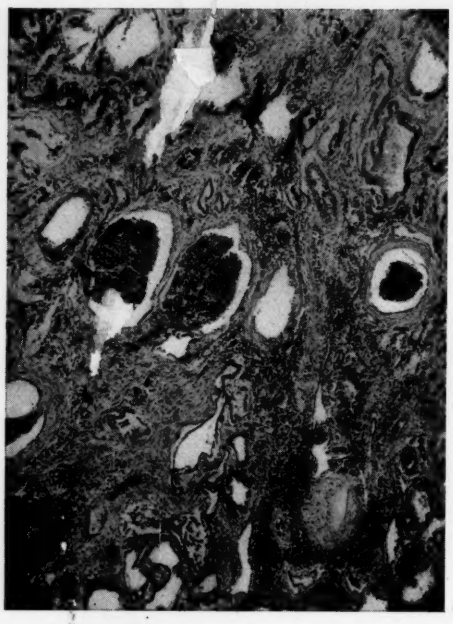

FIG. 389.—Microphotograph showing general renal destruction. The field was chosen to show the shrinking of the glomeruli and the dilated tubules, and represents one of the least affected portions of the kidney. (× 60.)

FIG. 390.—Microphotograph showing one of the larger calcific deposits in the interstitial tissue. In an adjacent degenerated tubule is a crystalline deposit of unknown constitution. (× 60.)

Another unusual feature is the presence of numerous small foci of calcification (*Fig.* 390). These are of varying sizes, the one depicted being one of the largest seen, and occur either in the connective tissue, or within or immediately adjacent to the basement membranes of the tubules. No calcification of glomeruli was noted, nor did there appear to be any calcific accumulations within the tubules, except where occasionally an apparent erosion of the cellular covering to a calcified deposit had left it protruding into the lumen. The assumption that these particles are calcific deposits is based on their staining reaction, their hard and gritty consistency, and the fact that the raised blood-calcium level, the parathyroid hyperplasia, and the experience of other observers, all provide a reasonable excuse for accepting their constitution to be that of salts of calcium.

In addition to these darkly-staining masses another type of foreign body is present in considerable numbers in the cortex. These consist of rounded bodies of peculiar appearance lying apparently within dilated lymphatics (*Fig.* 391). Their appearance is not unlike the conventional picture of the cross-cut of a tree trunk. They resemble, too, the soap plaques seen in fæces, and it is possible that they are indeed calcium soaps formed as an intermediate stage in the process of calcification. This suggestion is supported by the fact that in a few of them appear granules or streaks staining darkly with hæmatoxylin and representing

probably early foci of calcification. The smallest of these bodies appear immediately beneath the tubular basement membrane. Most of them occupy spaces in the newly formed fibrous tissue and are often surrounded by chronic inflammatory cells. They are insoluble in water and in the common fat solvents, and they are not doubly refractile.

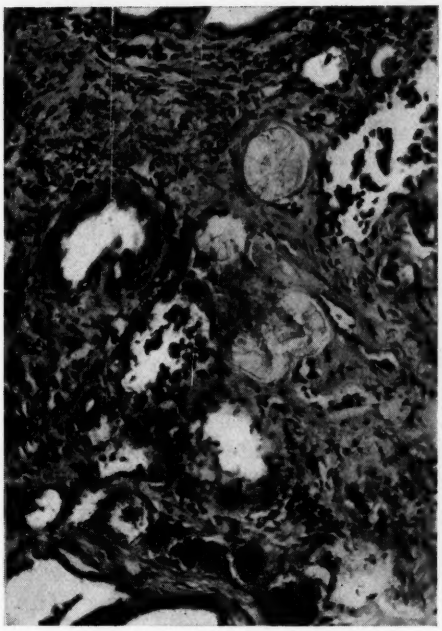

FIG. 391.—Microphotograph of a small collection of the interstitial and intralymphatic rounded bodies, which may be calcium-soap plaques. (× 60.)

DISCUSSION

The term renal rickets appears to have been first employed by Barber,[4] who published a description of ten cases in 1920, but there is no doubt that the association between stunted or deformed growth and chronic renal disease had been recognized long before this. Thus Lucas,[5] as long ago as 1883, reported a number of cases of rickets associated with albuminuria in late childhood, and used the term 'rickets of adolescence' to describe them. The credit of defining the condition more clearly as a clinical entity probably belongs to Fletcher.[6] Parsons[7], Teall[8], and Brockman[24] have added valuable contributions dealing with clinical and radiographic features, while theories on pathogenesis have been elaborated by Parsons[7] and Mitchell,[9] and more recently by Shelling and Remsen[10] and by Chown.[11]

CLINICAL RÉSUMÉ

A brief outline of the more usual clinical features may not be out of place at this stage. Although generally regarded as a disease first manifesting itself in late childhood, it is probably significant that a number of the patients have been reported as being unusually small at birth. The earliest symptoms usually occur about the age of seven, and consist of polyuria and polydipsia associated with a retardation of growth. The characteristic bony changes supervene in later childhood or may be delayed until after puberty, and it is on their account that the child is usually first brought under observation. There may be attacks of renal pain, especially

in those cases associated with secondary pyelonephritis or calculus, but these are by no means common features. The renal lesions, although invariably extensive at post-mortem examination, are, as a general rule, remarkably silent in their course.

The rapid development of genu valgum between the ages of ten and fifteen years is a rather frequent incident, and, occurring in a stunted child with a history of thirst and polyuria, should always suggest the possibility, or even the probability, of renal rickets. Thickening of bony extremities may be present, and subsequently, with increasing softening of the metaphyses, extreme degrees of distortion and deformity may be produced. Displacement of epiphyses is not uncommon, and an abnormal liability to fractures has been reported in several instances.

The majority of cases show little, if any, mental defect, but delayed sex development is common. The skin has a characteristic sallow or earthy tinge, the hair is sparse and dry, and a hypochromic anæmia is the rule. It is noteworthy that cardiac hypertrophy and vascular hypertension are quite exceptional in this disease, notwithstanding the fact that palpable thickening of peripheral vessels is frequently found. Ophthalmoscopic examination is negative in all except the rare cases showing hypertension.

The urine is copious and of persistently low specific gravity. The kidneys are incapable of excreting a concentrated urine. Some degree of albuminuria and cast formation is found in practically all cases. It may be associated in infected cases with pus cells and organisms. Renal function tests usually demonstrate gross inadequacy, and most cases terminate in uræmia.

BLOOD CHEMISTRY

The essential changes comprise azotæmia, lipæmia, and hyperphosphatæmia, usually, but not always, associated with a low calcium figure. There is in addition a lowered alkali reserve, or even, in some cases, an uncompensated acidosis. The blood-phosphatases are increased. The nitrogen retention may reach an extreme degree, a blood-urea or N.P.N. of over 200 mg. per 100 c.c. being quite a common finding.

An amazing feature of this disease is the feeling of comparative well-being which may coexist with such ominously high figures. It is difficult to realize that these patients live constantly beneath the shadow of uræmia, a catastrophe which is liable to supervene with dramatic suddenness, particularly after surgical intervention. In this connection it may not be inapposite to remark that the wise surgeon will set himself steadfastly against any such procedures, however loudly the deformed bones may seem to cry out for osteotomy.

RADIOGRAPHIC APPEARANCES

From a purely radiographic standpoint two quite distinct types of bone change are now recognized :—

Type A.—In this type the shafts of the long bones retain a reasonably normal density, but more or less florid rachitic changes are seen at the extremities differing in no material detail from those seen in ordinary rickets. There is no bending of the shafts, but simple displacement of epiphyses is liable to occur at the softened junctions in situations where they are normally subject to sheering stresses, such as the head of the femur.

Parsons[7] and Teall[8] state that it is possible for healing to take place so long as X-ray appearances conform purely to this type, but that healing never occurs in *Type B*.

Type B.—The appearances in this type are unique and quite unlike the picture associated with ordinary rickets. The bones show a general osteoporosis with loss of distinction between cortex and medulla. The cupped metaphysis is both broader and deeper than normal, and shows a ground-glass, woolly, or moth-eaten appearance with varying degrees of stippling. An appearance of subperiosteal erosion may extend for short distances along the diaphysis. Even the flat bones do not escape, and the vault of the skull shows considerable thickening. The osteoporosis here presents a peculiar honeycombed and stippled appearance reminiscent of the acute stage of Paget's disease or of certain cases of multiple osteitis fibrosa. These skeletal changes imply very severe metabolic disturbances, and Teall, who found them present in seven out of his ten cases, regards them as radiographically diagnostic of renal rickets. All the more severe deformities are found in association with this type, which for this reason has been simply termed the ' bending ' type. It must be emphasized, however, that it is not so much a bending of the shaft as a collapse of the softened metaphysis which gives rise to the really gross deformities. As Teall puts it, the metaphysis ' collapses sideways ', so that the epiphysis may be rotated through 45° or even 90° in a very short time. Secondary epiphysial displacement then occurs very readily, and may add considerably to the deformity.

In our opinion the peculiar radiographic features of *Type B* are probably to be correlated with parathyroid overactivity. It is now known that parathyroid enlargement is found in many of the advanced cases of the disease, and the resemblance to the skeletal changes produced by accepted hyperparathyroidism is too striking to be ignored. The significance of this will be developed in the later discussions on etiology.

The case here reported quite evidently conforms radiographically to *Type B* (*Figs.* 377–380), and reference has already been made to a considerable enlargement of the parathyroids discovered at autopsy.

REVIEW OF PATHOLOGICAL FEATURES

The pathological features of reported cases fall conveniently into three main divisions, viz., skeletal changes, endocrine abnormalities, and urinary tract lesions. Occasionally lesions occur in other systems, and some of these appear important in particular cases, but in the discussion which follows only the main lesions in these three systems will be considered.

Skeletal Changes.—The naked-eye and radiological appearances of the bones in the great majority of cases are sufficiently similar to those seen in rickets due to avitaminosis D to merit the name of renal rickets. In particular there is the marked enlargement of the epiphyses and the irregularity and broadening of the lines of ossification, and, as a rule, some degree of deformity of the long bones. The resemblance to ordinary rickets is, however, not borne out in all aspects of the bony change; in particular, the microscopical picture of the epiphysial line shows distinctly that the primary osseous and cartilaginous nature of the defective growth in rickets is here replaced by changes which appear to be secondary merely to excessive calcium resorption. While, as in infantile rickets, the line of ossification is irregular and twisted, and the junctional cartilage cells devoid of columnar arrangement,

there is here an absence of the usual rachitic proliferative changes in the cartilage cells. Also the bony trabeculæ present have little or no covering layers of osteoid, and the inter-trabecular spaces are filled with fibro-fatty tissue which may even extend into the spaces which should be occupied by marrow.

Shelling and Remsen,[10] Salvesen,[12] and others draw attention to the fact that the histological pictures of infantile rickets and osteomalacia on the one hand, and of hyperparathyroidism on the other, though usually quite distinct, may at times resemble each other to such an extent as to be indistinguishable. The microscopical picture seen in renal rickets is thus of little real value in solving the problem of its etiology, inasmuch as it indicates only that calcium depletion of the skeleton is the essential basis of the changes seen at the lines of ossification, without implicating exclusively either endocrine dysfunction or other disturbances of ossification.

The thickening of the skull seen in most cases is the result of soft and vascular overgrowth of cancellous bony spicules and of intertrabecular fibrous tissue, with loss of differentiation between the diploë and the outer and inner tables.

The Endocrines.—In general it may be said that the thyroid, adrenals, and pancreas show no evidence of abnormalities in these cases, and that the changes in the gonads have not been regarded as primary. The thymus in Shelling and Remsen's[10] case, as in ours, showed diffuse calcification of the Hassall's corpuscles and general atrophic changes; but among the reported cases it has never been considered to play anything more than a passive role. Of the remaining endocrines the parathyroids are so frequently involved as to be regarded by many as being as important as the kidneys themselves. The changes observed are simple hyperplasia of all the glands, or, less frequently, adenomatous enlargement of one or perhaps two of the glands. The histological appearances seen in these hyperplastic glands show no variations from the normal. The proportion of principal and oxyphil cellular elements appears unchanged, and the arrangements of the cells also show no abnormalities.

The pituitary gland has in a few cases been stated to show some minor abnormality of its anterior or posterior portions, but until quite recently it had never been seriously considered as causative of the condition. Chown[11, 13] has, however, put forward the theory that renal rickets is primarily the result of pituitary dysfunction. The implications of this hypothesis will be considered at a later stage. Meanwhile it may be stated that most of the pituitaries examined have shown little or no pathological change detectable by the means at our disposal.

The Kidneys.—As renal deficiency is the generally accepted basis of the disease entity under consideration, the picture presented is always one of some form of advanced renal destruction. In the majority of cases the kidneys are found to be small, contracted, and scarred, and histologically present the diffuse sclerosis and glomerular obliteration commonly found in cases terminating in uræmia. In a certain number of cases acute infective lesions in the form of a pyelonephritis may be superimposed, rendering difficult a proper appreciation of the pre-existent chronic renal changes. Occasionally the form of renal change is nephrotic in type, as in the case of Kass and Huenekens.[14] Even though in this case death did not result from renal failure the claim to the diagnosis of renal rickets seems well established, and it provides one of the aspects of this clinical entity which does not readily fit into the chemical theory of pathogenesis propounded by Mitchell.[9]

The observations of Ellis and Evans[2] and of others indicate that in certain cases the renal destruction is consequent upon some abnormality of the lower urinary tract of a congenital nature resulting in lesions such as hydronephrosis or dilatation of the ureters. In a small number of cases the renal lesion found has been congenital cystic kidney.

The microscopical structure seen in the kidneys in the different cases of renal rickets varies as would be expected from the great differences noted in the naked-eye appearances of the kidneys. Essentially progressive and advancing glomerular destruction is the only feature of note in all cases that have run their full course. As a general rule the arterial changes found appear to be secondary in type, and this is in keeping with the rarity of observed hypertension during life. An important feature observed in some cases is the evidence of calcuria provided by the presence of foci of interstitial and occasionally of intra-tubular calcification. Rarely calculi are found. As is to be expected, these cases usually coincide with those in which there is also parathyroid hyperplasia.

ETIOLOGY AND PATHOGENESIS

It has been found an extremely difficult problem to correlate the varied clinical and pathological features with any single etiological factor. One constant finding alone emerges from an examination of the accepted cases, viz., a pronounced degree of renal damage and deficiency in all advanced stages and in all post-mortem examinations. In a high proportion of cases this damage is effected by what Barber describes as " an insidious chronic interstitial nephritis of obscure etiology ". In a search for the cause of this obscure form of chronic nephritis specific infections such as syphilis and tuberculosis may be excluded at the outset, as also can be the acute infections of childhood, no one of which occurs with sufficient frequency to warrant its being singled out as possessing causative significance. It is also improbable that any of the ordinary forms of diffuse or focal chronic nephritis are constantly concerned, for in the great majority of these cases there is neither an antecedent history of acute nephritis nor any clinical or post-mortem evidence of hypertension or cardiac hypertrophy. The histological picture presented by the kidneys provides still further evidence that the process is an unusual one. Advanced glomerular destruction and gross tubular dilatation supply the usual evidences of renal failure, but the occasional indications of antecedent acute or subacute toxic nephritis, as provided by thickened and adherent tufts or by crescents in Bowman's capsules, are completely absent, as are also any evidences of a causative hypertensive arteriosclerosis. The only findings indicative of the possible cause of the renal destruction are suggestive of a true chronic interstitial inflammation such as is provided by chronic pyelonephritis. Similar progressive inflammation and fibrosis may conceivably be caused by the presence of any widely diffused chemical or physical irritant in the renal connective tissue. In the case here reported the densely fibrotic kidneys on section showed such a diffuse interstitial irritant in the form of minute calcific deposits. Such calcium deposition has been described in other cases, and attention has been drawn to the fact that analogous changes may occur in primary hyperparathyroidism. Much controversy rages over the possibility of some such purely endocrine causation of the observed renal lesion, and at this stage it may be advantageous to consider the two rival theories of the pathogenesis of renal rickets which at present hold the field. For the want of better names they may be called

the 'renal' and the 'endocrine' theories of origin, and the arguments which have been marshalled in support of each may now be considered.

The Renal Theory.—The renal theory of origin, briefly expressed, states that as the result of chronic renal disease in childhood there develops a condition of rickets with the attendant skeletal changes and deformities, and usually also with some degree of dwarfism and possibly infantilism. That renal deficiency usually terminating in uræmia is a more or less constant feature of the clinical syndrome has already been stressed, and it remains therefore to explain the manner in which the renal damage is believed to produce the skeletal changes. There are several modifications of the basic theory, but they all start with the inability of a damaged kidney to effect an adequate excretion of phosphates. The immediate result of this is a tendency to a raised phosphate level in the blood. The serum values of calcium and phosphorus are more or less complementary, and Parsons,[7] to whom the earlier conceptions of this theory are due, pointed out that by reason of the raised value of blood-phosphates a relative if not an actual deficiency of blood-calcium resulted. This relative calcium deficiency interferes with the deposition of bone, and results in an endogenous form of rickets.

Mitchell,[9] starting with the same premises, has considerably modified these views, and produced a theory which is the one generally accepted by the protagonists of the 'renal' hypothesis. According to his views, the fact that the unexcreted phosphates do not bank up progressively in the blood is explained by the fact that an alternative route of excretion is provided by the bowel. Mitchell believes that in the gut the excreted phosphorus may reach such concentrations as seriously to interfere with the absorption of the food calcium. Experimental evidence has been adduced in support of this theory. Thus rickets is readily produced in rats by a diet containing either high-calcium and low-phosphorus, or low-calcium and high-phosphorus, contents. It would appear that an excess of either one interferes with the absorption of the other. Similarly Orr, Holt, Wilkins, and Boone[15] have shown that in human infants excessive intake of either calcium or phosphorus interferes with the absorption of that element which happens to be in the lower concentration. Mitchell suggests that the excess phosphorus accumulating in the intestine by excretion acts in an identical way, fixes the food calcium as an insoluble calcium phosphate, and so leads to a true calcium deficiency rickets.

Theoretically such a calcium deficiency would appear to call for intensive administration of calcium by mouth in order to counteract any such discrepancy between calcium and phosphorus concentrations. In this connection it is of interest to note that Salvesen[12] claims to have benefited one of his cases materially by giving large doses of calcium lactate by mouth. The failure of ultra-violet light and of vitamin D concentrates to cure renal rickets also finds an explanation on Mitchell's theory, for as the calcium is already fixed no amount of vitamin will enable it to be absorbed.

These arguments afford a reasonable explanation of the bone lesions so long as a hypocalcæmia is present; but, as has already been noted, cases showing a normal or raised serum calcium are by no means rare. Figures as high as 15 or 16 mg. per cent have been obtained on repeated estimation in undeniable cases, and it is to be presumed that such hypercalcæmia is related to an increased activity if not to an actual hyperplasia of the parathyroid glands, particularly so as among the most recently investigated cases an increasing proportion of them do show

enlargement of some or all of the parathyroid glands. This hyperparathyroidism must be explained, and several suggestions have been put forward in this connection. One of the earliest of these presumes that a mobilization of calcium from the bones under the influence of the parathyroids is a necessary expedient to prevent the onset of tetany; but it is probable that this is not the whole truth. For one thing tetany is strangely uncommon in renal rickets, despite a calcium figure which sometimes falls as low as 4 mg. per cent. Actually a defence against tetany already exists in the form of an increased acidity of the blood. It is now recognized that calcium exists in the blood in two main forms, 'free' and 'bound', the latter being in loose combination with the plasma proteins. The ionization of the 'free' calcium depends on the *p*H of the plasma; the more this is lowered to the acid side, the greater the number of free calcium ions. The incidence of tetany appears to correspond rather closely with the reduction in the amount of ionized calcium present. In this connection it is interesting to note that by a method of ultra-filtration through collodion sacs, Pincus, Peterson, and Kramer[16] have shown that in chronic renal disease, although the total calcium in the blood may be much reduced, the reduction occurs mainly at the expense of the bound calcium, the free form being very little lowered. It may be presumed that a similar relationship between free and bound calcium occurs in renal rickets, and, as in addition the *p*H value of the blood often reveals an uncompensated acidosis, the comparative rarity of tetany, even when the total calcium is low, is easier to understand. On the other hand, the necessity for parathyroid hyperplasia becomes correspondingly more difficult to explain, especially when it is of sufficient degree to raise the calcium level to the high figures occasionally found. Actually it seems probable that too much attention has been focused on the blood-calcium, and that a more reasonable explanation is to be found in the hyperphosphatæmia. Admittedly hyperparathyroidism tends to correct a low calcium, but it also tends to correct a high phosphate level, and, as we have already shown, it is this retention of phosphorus which is mainly responsible for the metabolic embarrassment. Ellsworth and Howard[17] and also Shelling[18] have shown that administration of parathyroid principle causes mobilization and increased excretion of phosphate in man and animals, and it is probably to this end that the parathyroids undergo hypertrophy. On this view the hypercalcæmia is to be regarded as an almost accidental accompaniment, although admittedly unfortunate in the serious effect on skeletal stability with which it is associated.

Before leaving the renal theory it is to be noted that there are certain features of the syndrome of renal rickets which do not find a readily acceptable explanation on the basis of its arguments. It is pointed out by the opponents that the dwarfism, with or without infantilism, which is usually present, cannot easily be accounted for by chronic renal disease, particularly as these features are usually manifested in renal rickets long before the renal condition has progressed to the advanced stage at which they are likely to appear. On the other hand, these same features are very readily explicable on the basis of a primarily endocrine disturbance.

The Endocrine Theory.—The endocrine theory presumes a primary over-activity of the parathyroid glands, with or without an over-riding pituitary dysfunction. The former is sufficient to account for the skeletal demineralization and the deformities, but the dwarfism and infantilism are more obviously the effects of the pituitary. If it be admitted that the parathyroids are under pituitary control the problem is further simplified, for the whole of the mischief is then attributable

to the misdirected energies of this arch-dictator of the endocrines. The renal changes according to this theory are of a secondary nature. It is assumed that as a result of the hyperparathyroidic hypercalcæmia the kidneys will excrete calcium in excess. That calcuria does supervene in cases of hyperparathyroidism is proved, and it is suggested that in the process of calcium excretion some of the calcium reabsorbed from the tubules will accumulate in the peritubular lymphatics and tissue spaces, and there set up chronic simple irritative inflammation. This form of true interstitial chronic nephritis can be seen around calcific deposits in cases of osteitis fibrosa, and in many cases of renal rickets, including our own, a similar condition has been observed. That this interstitial inflammation can go on to destruction of the renal parenchyma seems undoubted, and thus the renal deficiency is fully accounted for as a secondary result of hyperparathyroidism.

Enlarged parathyroid glands have been reported in many of the recently recorded cases. Thus Shelling and Remsen reported hyperplastic enlargement of all four glands in their case, and they actually demonstrated, using Hamilton's[19] test, an increased content of parathormone in the patient's blood. Other cases, in addition to our own, have been reported by Smyth and Goldman,[20] Langmead and Orr,[21] and by Pappenheimer and Wilens.[22] That the calcific renal deposits found in these cases can account for the renal sclerosis usually present at post-mortem is suggested by some experimental observations. From the work of Albright[23] and others it is evident that extreme degrees of damage to the kidneys may result from the prolonged action of excessive parathyroid principle. In the experimental animal it is even possible by the administration of large doses of parathormone to cause complete anuria and death from renal failure in a few days.

Some of the arguments brought against this theory merit discussion. It is maintained that hyperparathyroidic skeletal demineralization produces a different radiographic picture from that of renal rickets. This is true of *Type A* renal rickets, but in the commoner *Type B* the pictures are often indistinguishable. In this connection it is noteworthy that *reported* cases of osteitis fibrosa in childhood are extremely rare. We would suggest that this may result from the fact that the skeletal deformities which develop in the growing bones resemble rachitic lesions so strongly that clinical attention is apt to be directed away from the real etiology of the condition, and that in fact these cases have been diagnosed as renal rickets. Another criticism levelled against the endocrine theory is that in large numbers of the reported cases there is no mention of parathyroid enlargement and/or calcific deposits in the kidney. That this is true of the great majority of the earlier reported cases is conceded; but examination of recent reports shows that these features appear in a large proportion of the cases in which search was made for them. The absence of renal calcific deposits may actually have little significance, as by the time the kidneys are examined they represent merely the burnt-out hulks in which a search for the cause of the fire which destroyed them can have little reasonable hope of success. That the renal calcific deposits are not present in much larger amounts is almost certainly due to the fact that the process of inflammation set up by them inevitably results in their partial or total removal.

A more serious objection to the endocrine theory is provided by the group of cases, already referred to, in which the renal damage is associated with congenital abnormalities of the urinary tract. Ellis and Evans,[2] describing twenty cases of

renal rickets, report the presence in fourteen of them of ureteric dilatations which are believed to be idiopathic and probably the result of a type of congenital neuromuscular imbalance at the ureteric orifices allied to the achalasias. Mitchell, in his large survey of cases, refers to a group of nine or ten in which renal damage was the result of congenital cystic kidneys. In other cases there have been other congenital lesions such as horseshoe kidney, ureteric stenosis, solitary kidney, etc. Such a high incidence of cases of these types does not accord readily with the endocrine theory, particularly when the congenital lesion is obviously sufficient in itself to account for the renal damage present. It may, however, be pointed out that, to those who accept the pituitary dysfunction theory wholeheartedly, such features as congenital abnormalities of the urinary tract find a rational explanation in an assumed disturbance in the adjacent diencephalic ganglia which are believed to control the development of the renal system.

Conclusions.—Such, then, are the two main theories and the arguments for and against each. It will be noted that each hypothesis is capable, with a little stretching, of accounting for most, if not all, of the features of renal rickets, and it behoves us to declare ourselves in favour of one or other of the two conflicting theories. Before attempting this it is essential that we define the syndrome of renal rickets. If we accept the definition of Shelling and Remsen, viz., " rickets resulting from chronic renal disease ", then obviously there is no argument as to which of the two theories is to be accepted. In fairness to these authors it should be pointed out that their definition, which so finally excludes any discussion as to the etiological nature of the disease, was made in the full acceptance of Mitchell's theory, and for the purpose of excluding cases of diverse types which had at one stage or another been included under the heading of renal rickets.

In our opinion the only possible definition of renal rickets at the present stage of our knowledge is one which makes no mention of etiology, but merely expresses the relationship of the principal features, e.g., " a disease of childhood characterized by skeletal demineralization with resultant deformities, and associated with chronic renal disease which in uncomplicated cases terminates in uræmia ". The acceptance of such a definition inevitably admits under the banner of renal rickets many cases which protagonists of the purely renal theory of origin would exclude, and raises the question whether the cases falling under this title do not perhaps belong to two, or even more, distinct disease entities. It must be clearly appreciated that the diagnosis of renal rickets is *not* made until both skeletal and advanced renal changes are present. It appears to us probable that the clinical condition diagnosable as renal rickets may be, not a disease entity of itself, but a syndrome common to either of two separate and distinct diseases in their late stages. Our discussion of the two theories of etiology of this syndrome has thus been directed towards explaining the pathogenesis of the two separate diseases, each capable of evolving a late phase which has been designated ' renal rickets '. The retention of the name ' renal rickets ' for this syndrome is in our opinion desirable, as not only is the condition a distinctive one, but the alternatives of classifying all our accepted cases as merely ' late sequelæ ' of hyperparathyroidism on the one hand, or of renal deficiency on the other, is not attractive. Justifiable diagnoses of renal rickets will therefore continue to be made, and although in many there will be little or no indication as to the parent condition from which they arose, there are nevertheless certain diagnostic ' pointers ' which may be of assistance.

At one extreme there is that group in which the primary disorder is endocrine. For our purpose it matters little whether we assign the major role to the parathyroids or to the pituitary. The skeletal changes are in any case to be regarded as the immediate result of a parathyroid overactivity, the effect on the sensitive growing metaphyses being especially severe, and producing a condition which in children may be indistinguisable from rickets. The hypercalcæmia which results from the extensive demineralization of the bones causes in its turn a progressive nephrosclerosis, terminating ultimately in renal failure. Clinically some degree of demonstrable bony change precedes the evidences of renal disease, so that any marked degrees of nitrogenous and phosphatic retention are late features. Collateral evidences of pituitary or diencephalic disturbances may be present, and in conjunction with the antecedent history may assist in the differentiation from the primarily renal group.

At the other extreme we have those cases arising as a result of congenital or other disturbances in the anatomy or physiology of the renal tract. Here the skeletal changes are secondary to the renal disease, and are to be regarded as the result of calcium deprivation, probably for the reasons suggested by Mitchell. Evidences of renal impairment appear early, and are associated with definite nitrogenous retention and with hyperphosphatæmia, while the blood-calcium remains low. When the metabolic disturbances reach a critical level, secondary parathyroid hyperplasia is evoked, and with the advent of this parathyroid overactivity the skeletal manifestations begin to alter. A progressive decalcification causes a general softening of the bones, and precipitates the characteristic metaphysial collapse which we know to play so important a part in producing many of the more grotesque deformities. Radiographically the picture which previously conformed to that of *Type A* renal rickets now takes on an unmistakable parathyroid imprint, and gives rise to the appearances recognized as *Type B*. Simultaneously there is a rise in the blood-calcium above normal levels, and secondary calcific deposits may form in the kidneys, blood-vessels, and elsewhere. From this point the onset of terminal renal failure is only a question of time. If it is known that albuminuria, cylindruria, or other evidences of renal trouble preceded clinical or X-ray signs of bone disease, and are associated with relatively low serum calcium and high phosphorus figures, the implications are that the case belongs to the renal group. Confirmatory evidence to the same effect will also be afforded if it is possible to demonstrate bilateral dilatation of the urinary tract or other congenital renal abnormalities by means of excretory urography.

It will be evident that with the appearance of severe renal damage in the first group and of secondary parathyroid hyperplasia in the second, a zone of overlap between two etiologically distinct diseases has been reached. It is here that the two pathways converge, and it is here also that the diagnosis of renal rickets is most frequently made. In many cases it will be extremely difficult to retrace with any certainty the steps by which this common ground has been reached. Such a difficulty confronted us in the case recorded in this paper. The hypercalcæmia and radiographic features pointed to hyperparathyroidism. At the same time there was such a degree of renal impairment that uræmia could not be long delayed. Little assistance was afforded by the history or by other clinical features, with the possible exception of the unusually long-standing polyuria and the fact that the dwarfism appeared to date from birth. These facts may be adduced as evidence

in favour of some form of pituitary or diencephalic disturbance, and the absence of any congenital renal lesion in the post-mortem findings (with due regard to their limitations) may be presumed to lend further support to this view. With these reservations we therefore conclude that our case should properly be assigned to the ' endocrine ' category.

SUMMARY

A case of renal rickets is described, with illustrations. The literature dealing with this condition is reviewed, and the clinical, biochemical, radiographical, and pathological features are summarized.

The ' renal ' and the ' endocrine ' theories of origin of the condition are critically discussed. The concept of renal rickets as a syndrome representing the common ground of overlap of hyperparathyroidism on the one hand, and of chronic renal damage on the other, is expounded and suggested as the most satisfactory in the present state of our knowledge.

We have much pleasure in acknowledging our indebtedness to Mr. E. W. Hey Groves, under whose care this case was admitted. We also have to thank the Medical Officer of Health for Bristol, Dr. R. H. Parry, and the Medical Superintendent of Southmead Hospital, Dr. P. Phillips, for permitting us to have access to the City Hospital records. To the latter, and also to Professor Bruce Perry, our thanks are due for their assistance in obtaining the post-mortem material, and for valuable suggestions. Finally we should like to thank Dr. Doris Stone and the University Department of Preventive Medicine for the biochemical investigations, and Miss D. Pillers for the coloured drawings and for her disinterested co-operation which enabled us to secure permission for the autopsy.

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CHOLESTEROSIS OF THE GALL-BLADDER: A REVIEW, SUPPLEMENTED BY PERSONAL OBSERVATIONS ON 87 CASES

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INTRODUCTION

The term 'cholesterosis' of the gall-bladder designates an interesting and fairly common condition in which the mucous membrane is infiltrated with a mixture of cholesterol esters and neutral fat. This lipoid material is distributed in a patchy fashion, forming bright yellow flecks of variable size, sometimes slender and scanty, sometimes distending each mucosal fold so that the gall-bladder seems to be lined with a thick, soft, golden, waxy fabric. The strands of this fabric run longitudinally, and terminate a short distance from the neck of the gall-bladder. Occasionally cholesterol polyps of mulberry form are present, and in some gall-bladders such are the sole site of gross lipoid deposit. The subserous coat contains an unusual amount of ordinary fat (*see Fig.* 403), and this fact may encourage the surgeon to essay a diagnosis of cholesterosis before the gall-bladder is opened. An excess of fat, however, frequently occurs in this site apart from cholesterosis. Gall-stones are found in about a third of all cases and are almost invariably of cholesterol-rich types. (*Figs.* 392, 393.)

The lipoid infiltration is finely dispersed within cells, both of the lining epithelium and of the supporting connective tissue, forming a sort of emulsion in the cytoplasm and producing a characteristic foamy microscopic picture. In the epithelium the lipoid may consist merely of a few globules in the basal parts of the cells, or, in extreme cases, the cytoplasm may be distended with droplets of varying sizes. The globules tend to be arranged in rows perpendicular to the free surface of the cells, and to be graduated so that the largest are next to the basement membrane (Policard, 1914). In the stroma the lipoid is contained in macrophages, which aggregate especially in the swollen tips of the plicæ. These aggregations may assume such proportions as to form the polypoidal masses mentioned above.

The story of cholesterosis is of recent growth, and the first reference to a condition of this nature was in 1857, when Virchow described a type of fatty infiltration of the mucosa of the gall-bladder, although he regarded the deposit as neutral fat excreted by the liver and reabsorbed by the gall-bladder. Aschoff (1906) observed the occurrence of cholesterol in the epithelium of the gall-bladder. Attention was directed afresh by Moynihan (1909) to yellow stippling of the mucosa, and it assumed a new importance owing to his terming it "a condition of the gall-bladder requiring cholecystectomy". Thereafter cholesterosis attracted the attention of surgeons and pathologists who previously had overlooked the condition, or who, like Virchow and Aschoff, had not regarded it as abnormal. Moynihan

was misled, however, regarding the nature of the lesion, for he believed the yellow flecks to be due to bile-staining of the submucous fibrous tissue as a result of desquamation of the epithelium from the tips of the plicæ. This error was repeated by MacCarty (1910), who coined for one type of the condition the now widely used name ' strawberry gall-bladder '. This attractive metaphor is apposite only to strawberries not fully ripe, but it is so picturesque that it has been translated into many languages. It has led numerous writers to suppose mistakenly that in cholesterosis the general background of the mucous membrane is dark red, and thence to make the specious deduction that cholesterosis is in essence a type of inflammation. Laroche and Flandin (1912) noted the association of cholesterosis with cholelithiasis. Lichtwitz (1914) went further and held cholesterosis to be an important contributing cause of cholelithiasis, for he believed that lipoid polypi, shed into the lumen, provide nuclei for gall-stone formation. This theory has received the support of many later writers (e.g., Gosset, Bertrand, and Loewy, 1928).

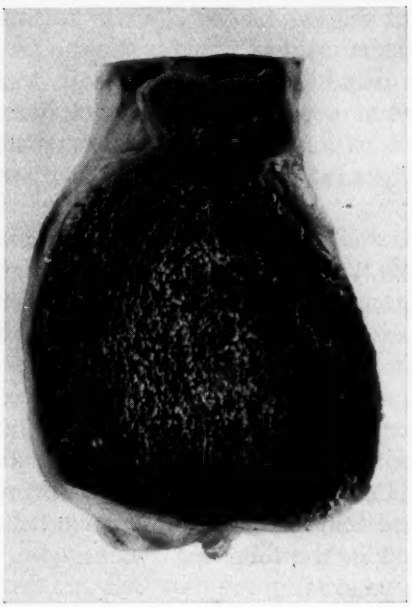

FIG. 392.—Cholesterosis of the gall-bladder. The plicæ of the mucosa are richly infiltrated with myelin fat, disposed as tortuous brightly-shining columns, in a general longitudinal direction. (*Natural size.*)

FIG. 393.—Cholesterosis of the gall-bladder with cholesterol-rich stones. The lipoid infiltration of the mucosa is abundant and characteristic. In this case the stones were recovered from the common bile-duct, having escaped down the wide cystic duct. The largest stone is a ' cholesterol solitaire ', the smaller ones are of mulberry type. Note the pigment centre of the split mulberry stone.

From the histological appearances of the gall-bladder epithelium Policard (1914) deduced that cholesterosis is due to resorption of cholesterol from the bile.

Boyd (1922 and 1923) showed that in cholesterosis the dried mucosa of the gall-bladder may contain as much as 60 per cent by weight of lipoid as against 0·6 per cent in normal controls, thus proving the condition to be a true infiltration. He showed also that the fatty material in the lipoid gall-bladder consists of cholesterol esters, and he was able to demonstrate these in practically all the tissues of the wall. Moreover, he found microscopic lipoid in about half of all gall-bladders examined. In the cholesterol gall-bladder he invariably found low-grade inflammatory changes, and concluded that these are the most important etiological factor, and Illingworth (1929), from an extensive pathological and clinical study, endorsed this view in the main, while pointing out that in some cases of cholesterosis evidence of inflammation is hard to find. Mentzer (1925) recorded some degree of cholesterosis of the gall-bladder in 37 per cent of all cases coming to autopsy at the Mayo Clinic. These figures and those of Boyd emphasize how extremely common is lipoid infiltration of the mucosa of the gall-bladder, but it is certain that in a large proportion of Mentzer's cases the amount of lipoid was quite small. Despite these observations indicating the extremely high incidence of some degree of cholesterosis, it has generally been regarded as a definite type of cholecystitis. Corkery (1922) is practically alone among later writers in suggesting that cholesterosis may not be a specific lesion but merely a random element in the protean manifestations of chronic cholecystitis.

It is commonly believed that the lipoid material is derived from the bile, from which cholesterol is resorbed by the activity of the lining epithelium of the gall-bladder. Elman, Graham, and Taussig (1931 and 1932), on the other hand, maintain that the gall-bladder secretes cholesterol, and that cholesterosis results when this secretory mechanism is impeded by low-grade inflammatory processes.

Despite much study and prolific writing many questions remain unanswered with regard to cholesterosis. Whence comes the lipoid—from the blood or from the bile? Is cholesterosis a manifestation of infective cholecystitis? Does it lead directly or indirectly to gall-stone formation? Does it of itself produce symptoms? It is the object of this paper to attempt to shed some additional light upon these problems, which will be discussed as indicated in the following synopsis:—

1. The Source of the Lipoid in Cholesterosis.
   - *a.* Experimental work on the cholesterol function of the gall-bladder.
   - *b.* The biliary cholesterol in human cases.
   - *c.* Resorption of substances other than cholesterol.
   - *d.* Blood cholesterol in man and animals.
   - *e.* Histological evidence regarding the cholesterol function of the gall-bladder.
2. Experimental Production of Cholesterosis.
3. Is Cholesterosis a Specific Form of Cholecystitis?
4. Gall-stone Formation in Cholesterosis.
5. Bacteriology of Cholesterosis.
6. Cholecystography in Cholesterosis.
7. Clinical Considerations.

1. THE SOURCE OF THE LIPOID IN CHOLESTEROSIS

Cholesterosis of the gall-bladder is essentially a form of xanthoma, arising as a result of the storage of a local surfeit of lipoid. This lipoid may be derived either from the blood or from the bile. If the source is the blood, then some

explanation must be sought for the great frequency of cholesterosis (*see*, e.g., Mentzer): why should the gall-bladder act as a magnet, so to speak, for cholesterol? Is the cholesterosis a manifestation of a cholesterol-excreting function normal to the gall-bladder? On the other hand, if the source be the bile, then the gall-bladder must be capable of resorbing cholesterol from the bile. In order, therefore, to clarify the question of the pathogenesis of cholesterosis it is necessary to solve the cognate question of the cholesterol function of the gall-bladder. There are three possibilities: the gall-bladder may secrete cholesterol; it may resorb it; or it may pursue the middle path of indifference. The problem may be attacked (*a*) experimentally, (*b*) by study of human cases.

a. **Experimental Work upon the Cholesterol Function of the Gall-bladder.**—Aschoff (loc. cit.), interested in cholesterol metabolism because of its bearing upon the formation of gall-stones, showed in a series of experiments on dogs that the gall-bladder mucosa frequently contains cholesterol esters histologically demonstrable. If, however, the cystic duct had been ligated beforehand, these esters could not be demonstrated, and so he concluded that the lipoid in the epithelium of the gall-bladder mucosa had its origin in resorption from the bile. He was unable directly to prove resorption of cholesterol, although he showed the resorption of neutral fats. He suggested, therefore, that normally the gall-bladder must resorb the cholesterol esters from the bile, and split these, returning the cholesterol fraction to the lumen. Blaisdell and Chandler (1927), by means of a diet rich in lipoids, were able to produce gross deposits of cholesterol in the gall-bladders of all of 10 dogs and 4 of 15 rabbits. Since ligation of the cystic duct prevented the appearance of these deposits, they concluded that the latter were the result of resorption from the bile. Torinoumi's (1923) experiments suggest that the normal gall-bladder of the dog takes up cholesterol from the bile, but if the gall-bladder be the seat of inflammation, either as an accidental result of operative interference, or intentionally from the introduction of typhoid bacilli, cholesterol will be poured out into the lumen, presumably with the exuded serum. Unfortunately these conclusions were based upon only three normal and two inflamed cases. Kusnetowsky's (1923) experiments dealt mainly with the large ducts, but also with the gall-bladder. It was shown that these structures readily took up fat and cholesterol esters introduced into their lumina. Illingworth (1929) demonstrated in the cat the disappearance of cholesterol from the lumen of the gall-bladder segregated by ligation of the cystic duct. He failed, however, to demonstrate it histologically in the wall of the gall-bladder, and suggested that in the gall-bladder, as probably also in the intestine, the cholesterol is taken up in a masked form which is not readily demonstrable with the polariscope. Loewy (1932) also concluded that the gall-bladder resorbs cholesterol. The most recent work on the cholesterol function of the gall-bladder is by Rousselot and Bauman (1935). These workers appear to have demonstrated without much doubt that the gall-bladder is capable of taking up cholesterol from a watery solution with bile-salts, and converting it into cholesterol esters.

Careful consideration must, however, be given to work published in recent years by Elman and Taussig (1931) and Elman and Graham (1932) suggesting that a function of the gall-bladder may be the excretion of cholesterol, i.e., directly the reverse of that mentioned above. Bile was subjected for varying periods to the activity of the mucosa of the gall-bladder which had been isolated by ligation of

the cystic duct. The amount of simple concentration that occurred was measured by comparing the concentration of bilirubin in specimens obtained by puncture at the beginning and at the end of the experiment. The concentration of cholesterol in each of these specimens was also determined. It was found that the cholesterol concentration increased more rapidly than the bilirubin concentration. Elman and Graham maintain that "since in these experiments bilirubin is indifferent to secretion or resorption by the biliary mucosa, its measurement furnishes a convenient gauge of changes in volume due to the action of the gall-bladder on the bile". If this premise be accepted, it is clear that increase of the cholesterol-bilirubin ratio indicates secretion of cholesterol, decrease of the ratio its resorption. The question, however, of the resorption or otherwise of bilirubin is one of the crucial questions upon which depends the correctness of the interpretation of these and many other experiments. It cannot be summarily dismissed in this fashion and it will be more fully discussed below.

More convincing evidence that cholesterol may actually be added to the bile during its sojourn in the gall-bladder is furnished by certain further experiments of Elman and Graham. The arrangement of the hepatic lobes and ducts in the dog renders it possible by appropriate ligations and cannulations to separate the biliary system into two districts, one of which (the central hepatic duct system) communicates with the gall-bladder. The bile from these two districts may then be collected separately, that from the central zone alone having been subjected to the activity of the gall-bladder mucosa. In the experiments under consideration the amount of liver tissue secreting into each duct system was determined approximately. In the specimens from each district an estimation was made of the amount of cholesterol present per gramme of liver tissue. It was found that in the specimen which had been subjected to the action of the gall-bladder, this was about twice as great as in the specimen coming directly from the liver. The authors concluded that cholesterol had been added in the gall-bladder.

These conclusions regarding the cholesterol function of the gall-bladder must, however, be scrutinized in the light of certain errors which are likely to upset the accuracy of such experimental work on the biliary tract. These are: (1) Bilirubin estimations are not a satisfactory control for cholesterol, since, as will be shown, bilirubin may be resorbed from the gall-bladder. (2) In experimental animals there frequently occur accessory bile-ducts, discharging directly into the gall-bladder, and through these cholesterol may be added to the contents of the latter despite ligation of the cystic duct. (3) As a result of trauma or inflammation, large amounts of cholesterol may be added to the contents of the gall-bladder by exudation of blood-serum.

The Inaccuracy of Bilirubin Estimations as an Index of Water-resorption Occurring in the Gall-bladder.—That the gall-bladder may not always be 'indifferent' to bilirubin is shown by the fact that even in the experiments of Elman and Graham the total amount of bilirubin definitely decreased as the experiments proceeded, and at the end of fifteen days it had disappeared. The brilliant work of Rous and McMaster (1921), which directed renewed general attention to the concentrating activity of the gall-bladder, also furnishes on re-analysis strong evidence of resorption of bile-pigment. It was shown in a series of dogs that when the gall-bladder is in communication with a system of ducts, from which discharge of bile is prevented by ligature, the gall-bladder and the ducts soon become filled with

dark bile thickened by addition of mucus. The highest concentrations of pigment were obtained, however, in experiments of quite brief duration, and in experiments lasting many days the amount of pigment in the ducts and also in the gall-bladder was considerably less. It may be argued that since the concentration effected by the gall-bladder is by a process of resorption of water and crystalloids which maintains the osmotic pressure of the gall-bladder bile equal to that of duct bile and also blood-plasma (Brand, 1902), the gall-bladder ceases to exercise an effective concentrating power as soon as the osmotic pressure of the less readily absorbed constituents of the contained bile approximates to that of the plasma. The fact, however, that the pigment content of the duct bile does not rapidly fall indicates that an important proportion of the concentrating activity of the gall-bladder is continuing; under these circumstances actual diminution in the pigment content of the gall-bladder surely strongly suggests resorption. Rous and McMaster note that disappearance of pigment may be masked for a time by conversion of bilirubin into the more deeply coloured biliverdin.

In certain of the experiments the biliary system of the dogs was separated by means of partitioning ligatures into a central area communicating with the gall-bladder, and a peripheral with the distal portion of the common bile-duct. So great was the concentration effected by the gall-bladder that, having to deal with only a fraction of the total bile secretion, it tended to collapse, and came to contain a small amount of dark viscid bile. This frequently contained less pigment than was to be expected from the area of liver drained by the central duct system, *but the total output of pigment remained constant*. Rous and McMaster suggest a selective inhibition of secretion in the central area of the liver, but it seems equally reasonable to assume that pigment was resorbed by the gall-bladder and re-excreted by the liver. Especially so since Aschoff (1909) demonstrated histologically resorption of bile-pigment by the epithelium of the gall-bladder. Further proof of resorption of bile-pigment by the gall-bladder is furnished by the absence of pigment from the contents of the hydropic gall-bladder encountered clinically. The gall-bladder becomes progressively distended with mucoid fluid and the pigment disappears. Graham has suggested that in some instances disappearance of bile-pigment from the gall-bladder may be the result of bacterial action, but in simple hydrops this may probably be excluded.

It is well known, of course, that in operative procedures upon the biliary tract of dogs a complicating infection is extremely difficult to avoid, and Riegel, Johnston, and Ravdin (1932) have shown by quantitative experiments that in the presence of infection 36 per cent of the bile-pigment may disappear from the gall-bladder in as short a time as two hours.

In view of all these facts, the conclusion is inevitable that, in animal experiments, repeated colorimetric estimations of bilirubin in the gall-bladder bile are quite unreliable as a standard whereby to judge the amount of concentration undergone by other substances in the bile. In other words, repeated calculations of the cholesterol-bilirubin ratio will not furnish accurate information as to whether cholesterol is being added to or taken up from the bile. If bilirubin is resorbed, then unless cholesterol be taken up at an equal or greater rate, the cholesterol-bilirubin ratio will inevitably rise. It is probable that the degree of water-resorption will be considerably underestimated; if this be so, the results of such calculations may fallaciously suggest that cholesterol has actually been added

to the contents of the gall-bladder. It is clear that the problem is capable of solution only by definitely quantitative experiments.

Is it Possible for Cholesterol to be Added to the Contents of the Gall-bladder Independently of Secretory Activity on the Part of the Mucosa?—Recent work of Andrews, Dostal, and Hrdina (1933) drew attention to the frequently ignored fact that the blood-plasma normally contains much more cholesterol than the hepatic or even the cystic bile. The figures vary, but the plasma cholesterol of the dog and of man is roughly 200 mg. per 100 c.c., while the gall-bladder bile normally contains 50–150 mg. per 100 c.c. Accordingly, in any experiments upon the cholesterol function of the normal gall-bladder, it is necessary to exclude the possibility of the exudation of serum into the lumen. In only one of Elman's experiments was the final concentration of cholesterol in the gall-bladder (271 mg. per 100 c.c.) greater than the accepted normal value of the plasma cholesterol in the dog, and in the remainder it was much lower. Any increase of total cholesterol, if such there were, might be explained as due to out-pouring of serum as a result of the trauma of operation. The highest reading could have been attained as the result of serous exudation supplemented by a slight exercise of the concentrating function of the gall-bladder.

Wilkie and Doubilet (1933) showed by experiments upon dogs that if the cystic duct be ligated, cholesterol may be added to the bile in the gall-bladder, or withdrawn from it, according as the concentration of cholesterol in the gall-bladder is less or greater than that in the blood-plasma. This implied in most cases a substantial addition of cholesterol to the gall-bladder bile. If, however, the cholesterol content of the gall-bladder bile had been artificially raised by direct addition of cholesterol to a value greater than the concentration in the plasma, then cholesterol was resorbed. In other words, in respect at least of its richness in cholesterol, the fluid in the gall-bladder came to resemble more closely the blood-plasma, the gall-bladder wall behaving as a membrane permeable to cholesterol. Wilkie and Doubilet state, however, that they were compelled to discard the majority of their animals because of inflammatory or gangrenous changes arising as the result of ligation of the cystic duct. The normality of the remainder is not therefore above suspicion, and so while these experiments are interesting as demonstrating the permeability of the inflamed mucosa, they cannot be accepted without reservation as throwing light upon the *normal* cholesterol function of the gall-bladder.

Some carefully controlled experiments upon the function of the gall-bladder with regard to bilirubin and cholesterol were made by Riegel, Johnston, and Ravdin (loc. cit.), utilizing dogs. A technique was employed which largely eliminated trauma to the gall-bladder; furthermore, since the experiments were quantitative, they avoided inaccuracies arising out of the use of the cholesterol-bilirubin ratio. A fine soft rubber catheter was passed up the common and cystic ducts into the gall-bladder, and the end of the catheter was brought to the exterior. A ligature was then applied around the central hepatic duct distal to the cystic duct. Damage to the cystic blood- and lymph-vessels was thus avoided, while the greater part of the bile continued to find its way into the duodenum. Their experiments dealt with an organ physiologically normal as judged by its ability to transform the hepatic bile of another dog into a fluid having the same composition as the bile which it had itself contained at the beginning of the experiment. Hepatic bile

was obtained from another animal, its cholesterol and pigment content determined, and a definite quantity introduced through the catheter into the gall-bladder. After a few hours it was withdrawn and changes in its volume and composition were determined. From these studies it was concluded that the normal gall-bladder does not resorb pigment, though this disappears rapidly if inflammation be present. In all of the uncomplicated experiments there was a slight decrease (10 per cent) in the cholesterol content of the gall-bladder, but where infection supervened the amount of cholesterol increased 96 per cent in twenty-four hours. Similar gross increases occurred when accessory hepatic ducts were present. The results clearly demonstrate that unless the occurrence of inflammation and the presence of accessory bile-ducts can be unequivocally excluded, no experimental evidence that the gall-bladder secretes cholesterol is admissible. The authors interpret their findings conservatively and incline to regard the 10 per cent diminution in the cholesterol content as within the limits of experimental error. The results, however, with regard to the cholesterol function of the gall-bladder are certainly more compatible with a resorptive than with a secretory process.

In conclusion, the balance of evidence obtained from the extensive series of experiments described indicates that the normal mammalian gall-bladder resorbs cholesterol from the bile. If, however, the gall-bladder be inflamed or traumatized, then effusion of blood-serum will greatly increase the amount of cholesterol in its contents.

b. **Evidence Based upon Estimations of Biliary Cholesterol in Human Cases.**—In a series of operations upon human cases of gall-bladder disease Elman and Graham took the opportunity of comparing the bile in the excised gall-bladder with that aspirated from the common hepatic duct during operation. The cholesterol-bilirubin ratio was determined in the two specimens of bile, and was found to be higher in the sample obtained from the gall-bladder. This was held to support their thesis that the gall-bladder secretes cholesterol, but it is clear that, before these estimations could be accepted as evidence, many fallacies would require to be excluded.

Chiray and Hesse (1932) analysed specimens of human bile obtained by duodenal tubage and found that the proportion of cholesterol to pigment was greater in the *B* specimen, derived supposedly from the gall-bladder, than in the *A* or *C* fractions which have their reputed source directly in the liver.

Accordingly, the small amount of evidence from human cases indicates that during the sojourn of bile in the gall-bladder, the concentration of cholesterol increases in greater measure than the concentration of bile-pigment. This, of course, falls far short of proof that the gall-bladder secretes cholesterol.

c. **Indirect Evidence from Experiments on Resorption of Substances other than Cholesterol.**—There has been much experimental work devoted to study of the ability of the gall-bladder to resorb a great variety of substances other than cholesterol and bile-pigments; these, being most closely germane to the question of cholesterosis, have been considered first. We cannot, of course, say that since the gall-bladder is able to resorb sodium chloride, therefore it also must resorb cholesterol, but as we shall see, the results of this great body of experimental work are more in keeping with resorption than with secretion of the latter. The difficulty of precise demonstration of the resorption of cholesterol contrasts sharply with the finality of experiments dealing with many other widely differing substances.

The water-resorbing function of the gall-bladder has been well recognized since the experiments of Rous and McMaster, and generally regarded as remarkable, though it is possibly not after all very extraordinary when it is compared with the daily performance of the analogous intestinal epithelium or the herculean labour of the renal epithelium. Demmel and Brummelkampf (1924) proved that the gall-bladder of the dog resorbs neutral fat, and Mentzer (1925) demonstrated fat in the epithelium of the gall-bladder of the rabbit twenty-five minutes after its introduction into the lumen. Whitaker (1929), using iodized oil, demonstrated it histologically in the epithelium of the gall-bladder of the cat and in the subjacent lymphatics, and showed that it may disappear in the course of a few weeks. Rosenthal and Licht (1928) demonstrated that the gall-bladder resorbs bile-acids, the disappearance of which could be accelerated by production of aseptic chemical inflammation. Iwanaga (1923), Mentzer (1925), and Nakashima (1926) showed that various types of dyes are rapidly resorbed. Nakashima found that the rate of resorption depends upon the diffusibility of the particular dye used rather than upon chemical composition, and Iwanaga demonstrated once again that the resorptive processes are accelerated by inflammation. Sweet (1929) found that sodium iodide in solution may disappear from the lumen of the gall-bladder within an hour, and Winkenwerder (1930) proved that ferrous ammonium citrate is very rapidly resorbed by the uninjured epithelium of the gall-bladder. Johnston (1931) showed that if sodium tetraiodophenolphthalein be introduced into the gall-bladder of a dog, 50 per cent may be resorbed in twenty-four hours. Andrews and Hrdina (1931) confirmed the findings of Mirovisch, Sachs, and Schrire (1930) that the gall-bladder has the power of resorbing calcium, and the latter group of investigators regarded this as so active a process that it might constitute a sort of internal cycle of calcium metabolism.

Secretion by the Human Gall-bladder.—When the gall-bladder is drained externally, the amount of fluid obtained depends upon the patency or otherwise of the cystic duct. If the latter be obstructed the daily drainage consists merely of a few c.c. of mucoid fluid. That the gall-bladder secretes mucus is well known. Phemister (1931) has shown that calcium carbonate passes into the human gall-bladder during periods when the cystic duct is obstructed by a stone, and may ultimately form a stucco-like mass filling the lumen. However, in gall-bladders containing this calciferous fluid (Kalk-milch-galle) the mucosa is usually the seat of advanced degenerative changes, and it may be difficult even to identity an epithelial layer, so that these observations do not establish the secretion even of calcium as one of the *normal* functions of the gall-bladder. Kerr and Lendrum (1936) have published an extraordinarily interesting case in which the presence in a gall-bladder of a papilloma composed of heterotopic intestinal epithelium was associated with profuse secretion of a fluid rich in sodium chloride. Such secretion has never been observed, however, in the normal gall-bladder.

The results of these extensive experiments and observations upon the physiology of the normal gall-bladder serve to establish beyond doubt that the activity of the latter with regard to a great variety of substances is resorption, and that the resorption is often accelerated by the hyperæmia and increased permeability of inflammation. If argument by analogy be permissible, it is reasonable to deduce that the normal function of the gall-bladder with regard to cholesterol will be resorptive also. This point will be discussed more fully later, from the histological aspect.

There is, it may be mentioned, a school of opinion represented by Sweet (1924), Blond (1927), and Halpert (1929), who have maintained that the normal gall-bladder does not merely concentrate the bile by taking up water, but actually resorbs all that enters during the inter-prandial period by the cystic duct, which functions as a one-way channel, and that it is at least unusual for any bile to be discharged from the gall-bladder down the cystic duct. Having once entered the gall-bladder the biliary constituents, including of course cholesterol, reach the intestine only after resorption, and re-excretion by the liver. Acceptance of this extreme view would clearly entail revision of all the accepted beliefs regarding the function of the gall-bladder and the mechanism of the expulsion of gall-stones from the gall-bladder and their passage along the ducts. None the less the ample evidence submitted of the very catholic resorptive capacity of the gall-bladder makes it appear at least possible that under certain circumstances the contained bile may be totally resorbed.

d. **Evidence from Blood Cholesterol Studies in Human Cases and in Animals.**—In the light of the accumulating evidence that only a small proportion of the cholesterol which daily passes out in the stool comes from the bile (Sperry, 1926; Sperry and Angevine, 1932), it is hardly to be expected that studies of cholesterol in the blood-plasma before and after cholecystectomy will show changes sufficiently marked to be of value in determining the role of the gall-bladder with regard to biliary cholesterol. In point of fact, the evidence is conflicting. Boyd (1923), using rabbits on a cholesterol-rich diet, found that following cholecystectomy the blood-cholesterol fell. Sweet (loc. cit.), on the other hand, found higher values in dogs after this procedure. Fowweather and Collinson (1926), studying the relation of hypercholesterolæmia to cholelithiasis in the human subject, found the average value of the plasma cholesterol in 11 cases to fall following cholecystectomy from 205 to 154 mg. per 100 c.c. If at all significant, the findings of Boyd and of Fowweather and Collinson would indicate that the gall-bladder resorbs cholesterol, those of Sweet that it secretes.

e. **The Cholesterol Function of the Gall-bladder in the Light of Histological Observations.**—The mucosa of the gall-bladder is lined by an epithelium of very uniform structure. It consists of a single layer of tall columnar cells, eosinophilic in reaction, with basally situated oval nuclei. Here and there a few goblet cells are scattered, relieving the simple monotony of the lining; in chronically inflamed gall-bladders they are much increased in number. The function of these goblet cells is beyond dispute—they are the source of a part of the mucus which is added to the bile during its sojourn in the gall-bladder, the remainder coming from well-formed muciniferous glands near the neck. The goblet cells therefore may be excluded from further consideration.

With regard to the function of the 'principal cells' of the mucosa, it may be observed that in the human body secretory and resorbing epithelia are sharply differentiated, and it is difficult to discover any instance of single epithelial cells that both secrete and resorb, although, e.g., in the intestine, cells of different types, some secretory, some resorptive, may be associated. In the gall-bladder lining, however, the outstanding histological feature is the structural homogeneity of the principal cells, and it is reasonable to assume that this connotes a corresponding uniformity of function representing in summation the normal physiological function of the gall-bladder lining. Now, as has been shown above, it is established that

under both physiological and experimental conditions this function is active resorption. The only known ' secretion ', apart from mucus, is calcium carbonate, which may accumulate in pathological gall-bladders when the cystic duct is obstructed. On the other hand, the *normal* gall-bladder is capable, under experimental conditions, of taking up large amounts of water, sodium chloride, calcium chloride, bile-salts, bile-pigments, fat, and a variety of ' foreign ' chemical substances. It is therefore prima facie unlikely that these cells, whose resorptive capacity is so generous, should possess in addition the power to secrete cholesterol.

In cholesterosis, abnormal amounts of lipoid may be found in the epithelial lining of the gall-bladder (*Fig.* 394), but characteristically by far the greatest

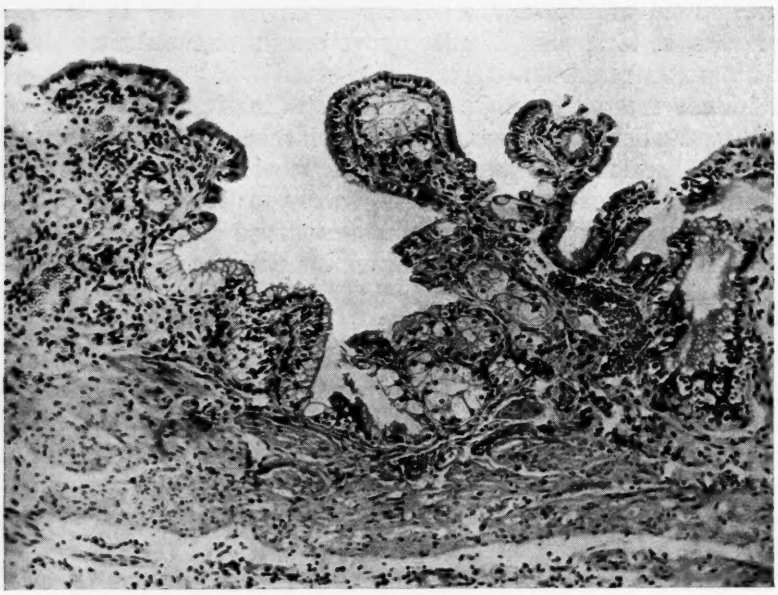

FIG. 394.—Cholesterosis of the gall-bladder. Paraffin section, stained hæmatoxylin and eosin. There is a rich deposit of lipoid material in the columnar epithelium of the gall-bladder, the cells of which in places are bloated and ' foamy '. In this specimen there is no stromal infiltration, although invaginated epithelial acini simulate nests of stromal macrophages. (× 100.)

amount occurs in collections of stromal macrophages (*Figs.* 395–400). These macrophages, elements of the reticulo-endothelial system, are scavenger cells, whose function is to segregate foreign substances, or normal metabolites present in excess of immediate requirements. In many pathological conditions they take up large amounts of lipoid, and if sufficiently numerous they form yellow masses, which may be either generalized or local, according as the lipoid excess is a widespread alteration in the composition of the body fluids, or represents merely a local accident. Generalized lipoid infiltration is exemplified by the lipoid histiocytoses, which are relatively rare—Gaucher's disease, Pick's disease, Hand-Schüller-Christian disease—and by xanthelasma and xanthoma diabeticorum, in all of which the basic lesion is an upset of lipoid metabolism and relative hypercholesterolæmia. Examples of localized deposit are provided by certain tumours, especially those of tendon-sheaths, and the osteoclastomata, by degenerating hæmatomata, cerebral softenings, certain cases of chronic pyosalpinx, and the

condition of fat necrosis of the breast. Cholesterosis comes definitely into the category of local lipoid infiltration, and accordingly the source of the lipoid excess

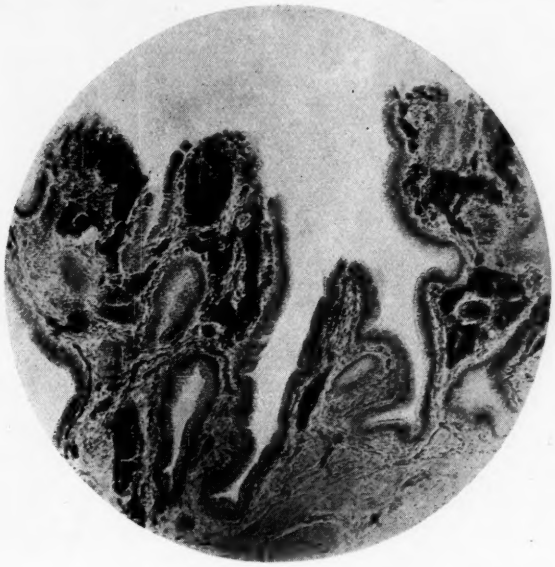

FIG. 395.—Frozen section of gall-bladder illustrated in *Fig.* 402, stained hæmatoxylin and scharlach R. The lipoid material occurs as darkly staining masses in the stroma of the plicæ. (× 75.)

FIG. 396.—The same section as illustrated in *Fig.* 395, photographed with a polarizing microscope. The doubly refracting lipoid is brilliantly shown and its fluid crystalline character suggested. In order to show the outline of the section the axis of the analyser has been set slightly off complete obscuration. (× 75.)

must be sought locally—and where if not in the bile? Now Illingworth has shown that in cholesterosis the bile in the gall-bladder may contain 967·5 mg. of cholesterol

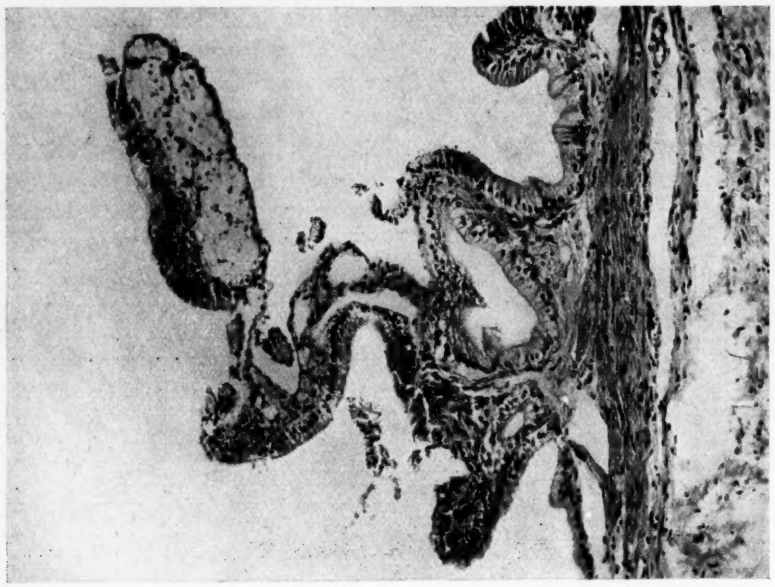

FIG. 398.—Cholesterosis of the gall-bladder. Paraffin section, stained hæmatoxylin and eosin. The histology is poor owing to delay in fixation. The specimen shows in the pedunculated bulbous tip of a plica a collection of macrophages solidly infiltrated with lipoid material. There is no trace of inflammatory infiltration, or of thickening of the wall of the gall-bladder. (× 100.)

FIG. 397.—Colour drawing of the frozen section of gall-bladder illustrated in *Fig.* 395. The lipoid material, stained orange, is contained in varying amounts in macrophages in the stroma of the plicæ. The stroma is cellular and appears œdematous, but there is no true inflammatory reaction. Red blood-corpuscles are stained green. (× 125.)

FIG. 399.—Cholesterosis of the gall-bladder. Paraffin section, stained hæmatoxylin and eosin. The mucous membrane is distorted by gross distension of the plicæ with masses of foam cells. This, however, is the sole departure from normal. There is no fibrosis no inflammatory infiltration, and no degeneration. (× 125.)

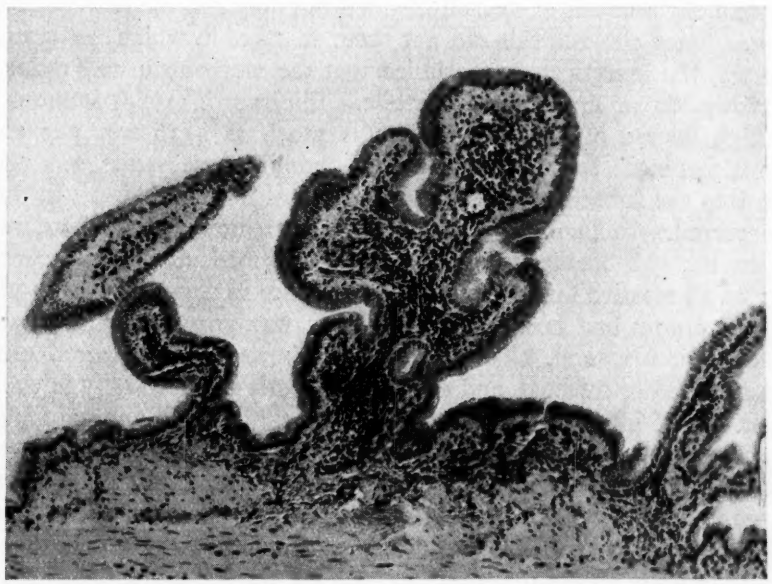

FIG. 400.—Cholesterosis of the gall-bladder. Paraffin section, stained hæmatoxylin and eosin. This gall-bladder contained stones. The wall is thickened, the muscularis is hypertrophied, and the mucosa heavily infiltrated with round cells. In the midst of the inflammatory tissue a solitary foam cell is seen standing out as a clear spot. (× 100.)

per 100 c.c., and if more evidence were desired that the bile in cholesterosis is unusually rich in cholesterol it would be furnished by the fact that in more than a third of the cases of well-marked cholesterosis, gall-stones of cholesterol-rich type are present. The gall-bladder resorbs as much as it can of the supersaturated cholesterol, yet it is unable to prevent crystallization.

Furthermore, the fact that the greater part of the lipoid deposit is in the stroma is hardly compatible with a secretory process. We do not find the secretion of a gland segregated in its stroma but in the cells which are producing the secretion, and in these cells the greatest amount and the largest globules occur near the free border. This is the direct reverse of the state of affairs in cholesterosis, which presumably therefore depends upon resorption.

2. EXPERIMENTAL PRODUCTION OF CHOLESTEROSIS

The ease of production of cholesterosis of the gall-bladder varies with the dietetic habits of the animal. Among herbivora, administration of gross excess of cholesterol leads to massive deposits in the liver, spleen, adrenals, aorta, heart, etc., and it is relatively difficult to produce lipoid infiltration of the mucosa of the gall-bladder. In the case of carnivora, on the other hand, it appears to be comparatively easy to produce a condition similar to human cholesterosis.

Dewey (1916) administered cholesterol to rabbits by intraperitoneal injection. Massive deposits were formed, especially in the liver and in the heart, and around some of these necrosis was noted, but in only a few cases was there the slightest evidence of lipoid infiltration of the mucosa of the gall-bladder. Blaisdell and Chandler (loc. cit.) used 15 rabbits and 10 dogs. For a prolonged period each animal received daily in its food 1 g. cholesterol suspended in egg-yolk. All the dogs showed cholesterosis of the gall-bladder, but only 4 of the rabbits developed this condition. Since cholesterosis did not occur in cases in which the cystic duct had been ligated, the investigators concluded that the mechanism underlying cholesterosis is resorption of lipoid from the bile. Illingworth (1929) induced in rabbits a very high degree of cholesterolæmia (as much as 1136 mg. per 100 c.c.) by cholesterol feeding. In 2 of the animals chronic cholecystitis was produced by injection into the subserous coat of streptococci of low virulence. In these cholesterosis occurred. In 4 controls receiving dietetic treatment alone there was no lipoid deposit in the gall-bladder. Patey (1934) performed similar experiments on 19 rabbits; in all massive lipoid infiltration took place in aorta, liver, and other organs. Where laparotomy had been performed there was gross lipoid infiltration of the healed abdominal wound. The gall-bladder, however, was spared unless it had been traumatized or infected, and the lipoid deposit occurred only in the scar tissue or at the site of inflammatory change. Thus the deposit appeared more usually in the subserous coat than in the mucosa. It occurred whether or not the cystic duct was ligated. The lesions, however, did not bear a very close resemblance to human cholesterosis. Patey concluded that, in his experiments at all events, the lipoid deposit was a xanthomatous condition dependent upon deposition from the blood, and that his results furnished no evidence regarding the debatable cholesterol function of the gall-bladder.

In the course of the work of Rousselot and Bauman (loc. cit.) upon the cholesterol function of the canine gall-bladder, a marked degree of cholesterosis occurred

following the introduction into the lumen for 48 hours in all of a 0·2 per cent solution of cholesterol in bile-salts. Increase in the cholesterol esters in the wall was accompanied by progressive and parallel disappearance of cholesterol from the contents of the gall-bladder.

3. IS CHOLESTEROSIS A SPECIFIC TYPE OF CHOLECYSTITIS?

During the past few years the author has studied histologically about 400 gall-bladders removed by various surgeons; among these 81 examples of cholesterosis were encountered, and in the majority the mucosal lipoid deposit was sufficiently abundant to be seen with the unaided eye. The figures probably exaggerate the incidence of gross cholesterosis, for many of the cases were discovered during a study limited to the less severe grades of cholecystitis; and cases of cholelithiasis were excluded from this series unless accompanied by cholesterosis (Mackey, 1934).

The general results of the histological examinations were, briefly, as follows: Lipoid infiltration occurred in the lining epithelium (*see Fig.* 394) or in the connective tissue of the mucosa, but more frequently and more abundantly in the latter (*see Fig.* 398). It was found in all types of gall-bladders; in some there was no evidence of inflammatory change; many were slightly thickened, and showed abundant infiltration with lymphocytes; while a few, especially those in which stones were present, were so greatly thickened and fibrosed that the lipoid infiltration constituted a very minor feature of the histological picture. In one extreme example of the last type the mucosa was crowded with densely aggregated lymphoid follicles and the lipoid deposit was confined to a few isolated macrophages within these follicles (*see Fig.* 400). The degree of cholesterosis was never proportional to the degree of inflammatory change, and was rare if the latter was severe. In short, the histological picture presented by cholesterosis is so pleomorphic that it is hard to believe that it is a pathological entity. Moreover, since accompanying inflammatory changes vary so greatly in degree, and may indeed be completely absent, the etiological basis of cholesterosis is surely not inflammatory.

The work of Cappell (1930) and others has shown that cells of macrophage type arise probably by differentiation of lymphocytes. These occur abundantly throughout the alimentary tract under normal conditions, and their presence in moderate numbers in the wall of the gall-bladder is probably not pathological. Similarly the foam cells should be regarded not as evidence of an inflammatory process, but simply as visible evidence of a process of lipoid resorption, common both to normal gall-bladders and to those showing moderate chronic inflammatory changes. It is quite likely, moreover, that cholesterosis may in some cases be transitory, disappearing should the biliary cholesterol fall to a lower level, or the gall-bladder wall lose its capacity to resorb and store the lipoid.

The cholesterol polypi which are so common in cholesterosis of the gall-bladder may be formed to accommodate a local superabundance of lipoid, or they may represent secondary cholesterosis of pre-existing polypi. Both mechanisms probably operate. In support of the second, it may be mentioned that polypi without lipoid are occasionally seen (*Fig.* 401), but they appear to be peculiarly susceptible to cholesterosis, for much more often the polypi contain lipoid when the remainder of the gall-bladder shows none.

Before concluding this discussion of the histological evidence bearing upon cholesterosis and the cholesterol function of the gall-bladder, it must be mentioned once more that Elman and Graham believe that the gall-bladder secretes cholesterol, and that this process becomes more active in the presence of inflammation. This, however, is unlikely, for in the ordinary course inflammation of a secretory epithelium leads to diminution or cessation of its secretion, not to intensification, and still less to the development of a new and highly specialized secretion. Nor

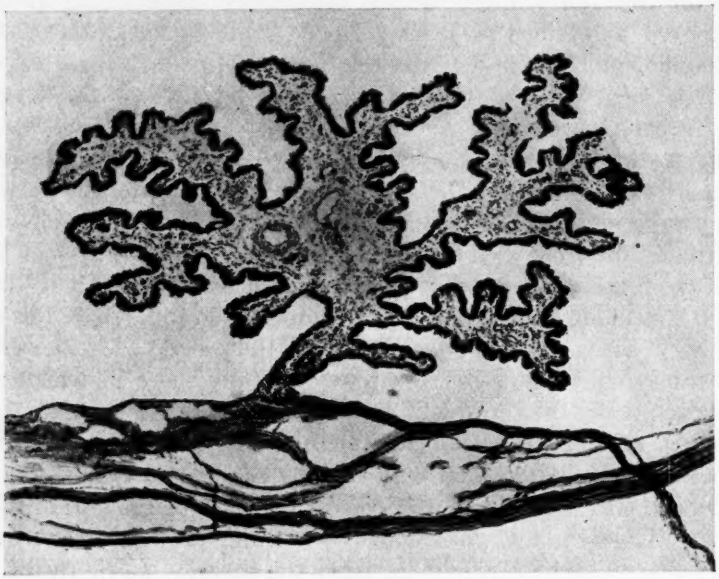

FIG. 401.—A polyp or papilloma occurring in a thin-walled degenerate hydropic gall-bladder. The polyp is covered with intensely staining short columnar epithelium. The stroma is well formed and vascular. There is no cholesterosis. (× 20.)

can it be admitted, for reasons given above, that cholesterosis is the direct result of an inflammatory process in the wall of the gall-bladder, and, as will be discussed in a later section, it is unusual to find organisms in the strawberry gall-bladder.

To sum up, cholesterosis is not a manifestation of cholecystitis, or of cholesterol secretion by the gall-bladder, but of active resorption of cholesterol, from bile unusually rich in that substance, by a relatively normal mucous membrane.

4. GALL-STONE FORMATION IN CHOLESTEROSIS

In a recent study of certain aspects of gall-bladder disease, 67 cases of cholesterosis were encountered, and of these 21 bore stones (Mackey, loc. cit.). There were 8 cholesterol solitaires, and 7 sets of mulberry stones. In 4 cases, cholesterol-rich stones had superimposed mixed laminæ, presumably of inflammatory origin; in only one case did the stones appear to be *primarily* of 'mixed' type. These figures correspond with the general experience. In about a third of all cases, cholesterosis is complicated by the presence of gall-stones. Almost without exception these are of cholesterol-rich types, cholesterol solitaires and mulberry stones occurring with practically equal frequency. Although the cholesterol solitaire is equally

common, the mulberry stone has been regarded by many writers as characteristic of cholesterosis, especially since Lichtwitz's original suggestion that its genesis may be traced to the dehiscence of cholesterol polypi. Without doubt these polyps, in gross form, are not unlike pedunculated mulberry stones, but the heart of a mulberry stone is composed of pigment, while the cholesterol polyp is a collection of macrophages containing cholesterol esters in foamy emulsion. A transformation of the one into the other is hardly conceivable. I have seen many hundreds of tiny mulberry stones in a gall-bladder bearing only a few small polyps, where it seemed scarcely possible that the latter could have given rise to so multitudinous a progeny.

FIG. 402.—Cholesterosis of the gall-bladder with pigment and mulberry calculi. The gall-bladder is normal in size and thickness. The mucous membrane shows heavy infiltration of the longitudinal plicæ with lipoid material. In the lumen there are four typical mulberry stones and a number of tiny pigment concretions.

FIG. 403.—External aspect of specimen shown in *Fig.* 402. There is abundant fat in the subserous connective tissue. The hyperæmia and the punctate hæmorrhages are results of operative trauma.

Moreover, when mulberry stones are numerous it is often possible to separate them into a number of groups, in each of which the individuals are uniform in size and presumably therefore of equal age. A theory implying that in these circumstances polypi have been shed in crops separated by fairly long intervals is, to say the least, highly speculative. The specimen illustrated in *Fig.* 402 is an unusually valuable piece of evidence upon the question of the origin of the mulberry gall-stone. It contained 4 typical mulberry stones with pigment centres and a superficial lamina of cholesterol, and in addition a crop of tiny spicular pigment stones, some of which if retained in such a gall-bladder would surely form the nuclei of the succeeding brood of mulberry stones. The Lichtwitz theory is attractive but untenable, and, on the whole, the smooth radially arranged mulberry stone with its soft pigment

centre is best explained as the end-result of a special type of cholesterol crystallization, upon an irregular pigment nucleus. Illingworth (1936) has recently expressed a similar opinion.

Rovsing (1925), indeed, traces the origin of all gall-stones, even the cholesterol solitaire, to crystallization of cholesterol upon nuclei of 'bilihumin', one of the less soluble bile-pigments. This he insists is not a constituent of normal bile, but is produced in the liver in diseased and toxic states, and frequently during pregnancy. In the bile capillaries it forms irregular concretions, which are swept down by the bile, often presumably in showers, to the larger ducts and the gall-bladder. It is certain that the vast majority of all gall-stones have a kernel of pigment—even on occasion the cholesterol solitaire. Some solitaires do not have a pigment nucleus, and Rovsing suggests that the pigment originally present has disappeared by diffusion. It may well be, however, that the solitary cholesterol stone may arise as a result of simple crystallization from bile containing a gross excess of cholesterol. If at a time when the gall-bladder bile becomes supersaturated with cholesterol, the gall-bladder happens to contain a few pigment concretions, the cholesterol will naturally separate out on their surfaces, and give rise to mulberry calculi. New broods of stones are laid down each time the arrival of pigment nuclei in the gall-bladder coincides with the occurrence of an excess of cholesterol in the bile. When the stones become numerous and occupy the greater part of the lumen of the gall-bladder, they become faceted (cf. *Fig.* 404).

FIG. 404.—Cholesterosis of the gall-bladder with three large cholesterol-rich stones. The lipoid deposit is moderate in amount, and it has been rendered less prominent by lytic changes following removal of the gall-bladder. The three stones are of considerable age. They illustrate how, when stones come to occupy the greater part of the cavity of the gall-bladder, facets form on contiguous surfaces.

In view of the frequency with which we find the cholesterol solitaire in the strawberry gall-bladder it is quite clear that there is no specific connection between cholesterosis and mulberry gall-stones. The latter are merely cholesterol-rich stones whose character has been determined by the shape of their pigment nuclei, by the number of these nuclei in relation to the size of the gall-bladder, and by the complex variations in the character of the mother-liquor. Cholesterosis might equally well occur in association with multiple faceted stones were it not that, generally speaking, the latter, by their very numbers, have set up pathological changes in the wall of the gall-bladder so severe that it has lost the capacity to develop cholesterosis.

It is generally accepted that the 'solitaire' calculi, so frequent in cholesterosis, arise aseptically in non-inflamed gall-bladders by crystallization from bile supersaturated with cholesterol. This is in itself an argument against the existence of an inflammatory basis for cholesterosis.

To conclude, cholesterosis and lithiasis are cognate manifestations of supersaturation of the bile with cholesterol, not cause and effect. Cholesterosis, however, is not without important significance, for it certainly betokens a metabolic state with an unduly high level of biliary cholesterol, and likely therefore to lead to gall-stone formation. The etiology of the characteristic stones, both 'solitaire' and 'mulberry', is primarily metabolic, not infective, although inflammatory complications of mechanical origin are likely to supervene later.

5. THE BACTERIOLOGY OF THE GALL-BLADDER IN CHOLESTEROSIS

On this point published figures are few. Illingworth reported 35 cases, 17 with stones. Of this total 29 proved sterile, 3 harboured streptococci of avirulent types, and 2 coliform bacilli. In the course of a recent bacteriological investigation by the author of 100 unselected gall-bladders removed at operation by various surgeons, the gall-bladders were dissected under aseptic precautions, and cultures, aerobic and anaerobic, on solid and fluid media, were made from the various layers of the wall, from the cystic lymph-node when this was present, and from the bile. In the 14 cases of cholesterosis encountered in this series the absence of infection was very striking, for in 13 no growth was obtained from any of the materials examined, and in the remaining 1 a non-hæmolytic streptococcus was found. It may in fact be said that in simple cholesterosis the bacterial flora of the gall-bladder is not more rich than that of gall-bladders which, on histological grounds, would be regarded as practically normal.

6. CHOLECYSTOGRAPHY IN CHOLESTEROSIS

According to Rossi (1932), who, incidentally, believes that cholesterosis is a pathological manifestation and is accompanied by symptoms, the cholesterol gall-bladder may be visible in the direct film of the upper abdomen. He states further that following administration of the cholecystographic dye the shadow appears slowly, is ill-defined, and may be mottled; after a fatty meal it disappears quickly, often with an exacerbation of pain. So far as the author knows this association of pain with cholesterosis has not been confirmed. Illingworth (1929) found that in the absence of other evidence of definite cholecystitis, the cholesterosis gall-bladder produces a dense normal shadow, and that contraction is active following the fatty meal. The author's observations previously reported (loc. cit.) may be reproduced here in tabular form (*Table I*). They concern 58 cases of cholesterosis, 20 complicated by the presence of stone.

Table I.—Cholecystographic Response in Cholesterosis

Type of Gall-bladder Lesion	Total Cases	Normal Density	Faint	No Shadow
Cholesterosis	38	9	25	4
Cholesterosis with stone(s) ..	20	0	17	3

These figures demonstrate that, in the absence of stones and of secondary cholecystitis, the gall-bladder showing cholesterosis is frequently capable of achieving a normal concentration of bile and dye. The rapid diminution in size after the fat meal mentioned by Rossi and Illingworth indicates either brisk contraction of the gall-bladder musculature, or, as Halpert, Sweet, and Blond would have it, rapid resorption of the contents of the gall-bladder for re-excretion by the liver. In any case it betokens a gall-bladder functionally not far removed from normal.

7. CLINICAL CONSIDERATIONS

It has never been possible to associate with cholesterosis a pathognomonic symptom-complex; the complaints attributed to it have always been those generically termed 'cholecystitic'. In some cases there has been a history of biliary colic, in others of 'gall-bladder dyspepsia'. Such symptoms, however, occur in all types of derangement of the biliary apparatus, both functional and organic, and probably some of them may occur also in upsets of other parts of the alimentary tract. Reports of clinical results following cholecystectomy have been conflicting, and this is not surprising, since, as has been shown, cholesterosis is a histological feature that may be found in gall-bladders otherwise normal, or in association with lesions in all degrees of severity.

Moynihan's paper, "A Disease of the Gall-bladder Requiring Cholecystectomy", has exerted a considerable influence upon subsequent surgical practice. He had been accustomed to perform simple cholecystostomy in cases presenting symptoms referable to the gall-bladder but in which at operation no gall-stones were found. Six cases were recorded in which this treatment produced no benefit, and which were subsequently relieved of their pain by cholecystectomy. In each the mucosa of the gall-bladder was marked by yellow stippling, and the coincidence seemed so striking that Moynihan attributed the pre-operative symptoms to this abnormality. It has been shown, however, that true biliary pain will be cured in a large proportion of cases by cholecystectomy, even if the gall-bladder be practically normal; it is not correct, therefore, to assume that the symptoms in these cases were the result of the presence of cholesterosis, and still less that cholesterosis invariably produces painful symptoms. The correct interpretation is simply that biliary pain, unrelieved by cholecystostomy, may in some cases be cured by cholecystectomy. Judd and Mentzer (1927), in a study of 1000 cases of cholesterosis treated by cholecystectomy, found that good results were achieved only if pain had been a dominant symptom. White and Riddick (1928) concluded that cholesterosis is only part of a general metabolic dysfunction, and that cholecystectomy is therefore unlikely to lead to relief. Stanton (1932) had 6 cases of cholesterosis in his series of 93 stoneless cases, and of these 4 were not benefited by cholecystectomy. On the other hand, Young (1928) had 45 strawberry gall-bladders in his large series, and formed the impression that "some of the worst cases, with a marked tendency to myocardial degeneration, were strawberry gall-bladders, and some of the most marked relief was achieved after removal of these". Such occurrences are among the happy mysteries of biliary surgery. From time to time one encounters reports describing relief of rheumatoid arthritis, myocarditis, or nephritis following removal or drainage of a gall-bladder which showed no very gross lesion, or possibly contained a cholesterol solitaire. Kopp (1929) alleviated

by cholecystectomy the symptoms of 7 out of 11 cases of cholesterosis without stone. Gosset (1928) reported improvement in 13 of 16. Illingworth (1929) could trace only 6 of his 35 cases, and these over a relatively short period, but all appeared to be well.

In a recent investigation, already quoted, of the end-results after cholecystectomy in minor degrees of gall-bladder disease, the post-operative histories were traced of 52 cases of cholesterosis, 19 with and 33 without gall-stones (Mackey, loc. cit.). These are summarized in *Table II.*

Table II.—The Clinical Result after Cholecystectomy in Cases of Cholesterosis

Type of Lesion	Total	Well	Improved	Unimproved	Post-operative Death
Cholesterosis without stone ..	33	14	2	16	1
Cholesterosis with stone(s) ..	19	6	13	0	0

All the cases with gall-stones were at least improved after removal of the gall-bladder, but of the stoneless cases half were unbenefited. The results in this latter category were no more satisfactory than those in a larger series in which the histological changes in the gall-bladder were minimal. The end-results of cholecystectomy for gall-stones will always be better than those in ' cholecystitis without stone ', for in a proportion of the latter the pre-operative complaints have not had their origin in the gall-bladder. In this connection it should be emphasized that cholesterosis *per se* probably does not produce symptoms, and certainly none likely to be relieved by cholecystectomy. Further, the end-results shown above were closely related to the character of the pre-operative complaints. Patients who had suffered before operation from true gall-bladder colic were almost always improved, but in those with more vague complaints the outcome was often unsatisfactory. It is probable that true biliary colic may occur, even apart from organic disease, in functional derangements of the gall-bladder (Westphal, 1923), and that in most of these cases it will be relieved by cholecystectomy. On the other hand, it is quite likely, in cases with vague dyspeptic symptoms, that the latter have had their origin outside the gall-bladder. Consequently, little benefit is to be expected from cholecystectomy in such indefinite cases. Subjective symptoms are of course by no means proportional to the severity of gall-bladder lesions, and depend greatly upon the psychological constitution of the patient.

In view of the frequency with which gall-stones complicate cholesterosis the suggestion may be made that when the gall-bladder is the seat of this lesion it should be removed, in order to *prevent* gall-stone formation. This is surely an undesirably radical and dangerous form of treatment for a condition that appears to be merely an unusual manifestation of a normal physiological process.

It is clear, however, that to discuss the surgical treatment of cholesterosis *per se* is really beside the point, for the diagnosis of cholesterosis cannot be made until the gall-bladder is exposed at operation and opened. If this operation has been dictated by typical gall-bladder symptoms, such as recurring biliary colic, or

if at operation gall-stones are found, then the correct treatment is cholecystectomy, whatever the condition of the gall-bladder mucosa. If, on the other hand, symptoms have been vague or atypical, and the operation is in a sense exploratory, then the mere presence of uncomplicated cholesterosis neither calls for, nor justifies, cholecystectomy, for there is no evidence that it is ever responsible for the production of symptoms.

SUMMARY AND CONCLUSIONS

A review is presented of the subject of cholesterosis of the gall-bladder, supplemented by personal observations of 87 cases.

'Cholesterosis' is the term applied to a common condition in which the mucosa of the gall-bladder is infiltrated with lipoid substances. In its nature it is related to the xanthomata.

It is not a specific lesion, but rather a histological feature that may occur at random in gall-bladders showing all grades of pathological change; in fact, apart from lipoid infiltration, many are normal. In the presence of advanced inflammatory or degenerative changes, cholesterosis is rare.

It is probable that the gall-bladder has the power of resorbing cholesterol from the bile. When the cholesterol is unusually abundant the process may become, as it were, visualized, and cholesterosis result. Should the amount of cholesterol in the bile diminish later, the cholesterosis probably may disappear.

The gall-bladder showing simple cholesterosis rarely harbours a demonstrable organismal infection.

In a third of the cases of cholesterosis, gall-stones occur; these are almost always of cholesterol-rich types. The cholesterosis and the stones are not respectively cause and effect, but are parallel results of super-saturation of the bile with cholesterol. Cholesterosis betokens a state of affairs in which gall-stones are liable to be formed, but it is not otherwise of clinical significance.

Cholesterosis does not, *per se*, produce symptoms. Consequently, the mere presence of lipoid infiltration of the mucosa of the gall-bladder does not justify cholecystectomy, nor does it support a prediction that the clinical result of this operation will be gratifying.

Acknowledgements.—This paper is principally the result of work begun in 1933 at St. Louis, when the author was working under the guidance of Professor Evarts A. Graham. It was at his suggestion that the study of cholesterosis was undertaken, and to him warm thanks are due for clinical and pathological facilities unstintingly furnished, and not less for his sustained and stimulating interest in the investigation, an interest unimpaired by the fact that the author's views on the cholesterol function of the gall-bladder were, and are, at variance with his own.

Some of the early pathological material was obtained in the Pathological Institute of the Western Infirmary, Glasgow. The three specimens illustrated by Miss Brown Kelly's admirable colour drawings are in the Museum of the Department of Surgery, University of Glasgow. For the use of these and for his interest and encouragement I have to thank my chief, Professor Archibald Young.

The photomicrographs (*Figs.* 397, 398, 400, 401, 403) and the photograph (*Fig.* 392) are by Mr. John Kirkpatrick. This last is from an illustration in Sir Robert Muir's *Textbook of Pathology* (Edward Arnold).

I am indebted to Professor Browning for a great deal of valuable constructive criticism during the writing of the paper, and to Miss A. M. Kelly for her excellent secretarial work.

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VISITS TO SURGICAL CLINICS AT HOME AND ABROAD

PROFESSOR GREY TURNER AT THE BRITISH POST-GRADUATE MEDICAL SCHOOL

THE foundation of this institution, which is the first of its kind in this country, marks a notable epoch in the history of British medical education. The hospital which affords its clinical basis is a modern building which will contain 534 beds when certain additions are complete. It was originally built as a poor-law hospital, and had hardly been completed in 1914 when it was taken over temporarily by the medical department of the War Office, and then used as a special military orthopædic hospital, where Sir Robert Jones had the centre of those training and organizing activities which were of such inestimable benefit to our wounded in the War. It was transferred in 1929 to the London County Council, and represents the most recent of all the many hospitals controlled and administered by that body. It is situated in Ducane Road, Shepherd's Bush, Hammersmith, on the westerly fringe of greater London. The idea of a post-graduate medical school was first initiated by the Right Hon. Christopher Addison in 1921 when he was Minister of Health. After long and careful consideration of various possibilities it was decided to found the new school under the ægis of the London University in affiliation with the London County Council hospital at Hammersmith. This, too, marks a very significant development in the hospital policy of this country, viz., the assumption of the highest type of clinical research and training by a municipal hospital in association with a University. So that already this Hammersmith hospital has made history both during the war and now in the initiation of post-graduate teaching.

The medical school, whose charter was received in 1931, was formally opened by King George V on May 13, 1935. The teaching work is divided up into five departments: Medicine, Surgery, Obstetrics with Gynæcology, Pathology, and Radiology. The staff, apart from consultants called in occasionally, is divided into two categories: the one consists of whole-time professors, readers, and assistants, and the other of selected teachers from other medical schools who give lectures and clinical courses on special subjects from time to time. The permanent staff of the present surgical unit is as follows:—

Director: Professor G. Grey Turner.
Reader: Mr. A. K. Henry.
Senior Assistant (*L.C.C.*): Mr. D. L. Lewis.
First Assistant (*Orthopædics*): Mr. A. J. Watson.
First Assistant (*Genito-urinary*): Mr. A. L. Light.
First Assistant (*Genito-urinary*): Mr. G. Y. Feggetter.
First Assistant (*Gastro-enterology*): Mr. R. H. Franklin.
Assistant (*E.N.T.*): Mr. J. I. Griffiths.

For the current session the following teachers are giving special lectures and demonstrations :—

Mr. Geoffrey Jefferson : *Surgery of the Nervous System.*
Professor John Fraser : *Acute Bone Infections.*
Professor Seymour Barling : *Other Acute Surgical Infections.*
Mr. W. B. Gabriel : *Rectal Diseases.*
Professor Hey Groves : *Fractures.*
Mr. J. E. H. Roberts : *Surgery of the Chest.*
Mr. Russell Howard : *Diseases of the Breast.*

FIG 405.—An air view of the Post-graduate School and Hospital.

It would be outside the scope of the present article to attempt any detailed description of the buildings. The medical school, which is joined to the hospital, contains a large out-patient department, which is also used on one or two days each week as a fracture clinic ; two operating theatres, with attached rooms for sterilizing and anæsthesia ; a very extensive unit of pathology, including an animal experiment department, museum, and post-mortem room ; a library ; and an X-ray department for diagnosis and treatment. (*Figs.* 405–408.)

It was appropriate that the first official visit of the Moynihan Surgical Club to London on Oct. 9 and 10, 1936, should have been to the clinic of Professor Grey Turner, because he was one of the original members of the club, which he had entertained previously at Newcastle-on-Tyne, before his migration to

London, where he holds the onerous and responsible post of the first Director in Surgery of the new British Post-graduate Medical School.

Having been welcomed by the Dean of the School, Colonel A. H. Proctor, D.S.O., I.M.S., we began our visit by watching Prof. Grey Turner do three operations. Before each operation he gave a short discussion, together with a typed history of the patient's illness.

Case 1. A Girl of 5 years with Extroversion of the Bladder.—The child had great dilatation of both ureters, highly infected urine, and had had several febrile attacks, which had not been relieved by ureteral catheterization. At a previous operation the right kidney had been drained, with very marked improvement of the

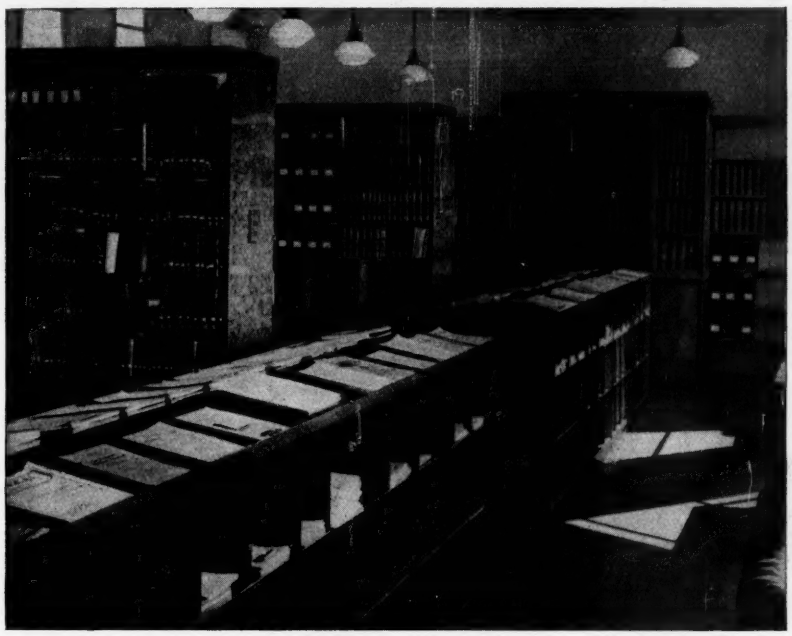

FIG. 406.—The library.

general condition. The abdomen was opened by a midline incision. Both ureters were found to be greatly dilated to a diameter of about 1 in., the right being slightly the larger. The presacral nerve was exposed and resected, with the object of reducing the dilatation of the ureter. It was not considered wise to attempt to transplant the ureter at this stage, and the operation concluded by drainage of the pelvis of the left kidney. The patient stood the operation very well and did not appear to be appreciably upset.

Case 2. A woman of 60 with Recurrent Gall-stone Attacks.—Gall-stone attacks since 1917. In 1931 she suffered from acute cholecystitis, for which the gall-bladder had been drained after removal of the stones. Since April, 1936, she had suffered from recurrent attacks of colic and vomiting, and during each attack she could feel a lump at the site of the operation scar, and she was mildly jaundiced. In August, 1936, she had an abscess of the gall-bladder pointing; this was incised,

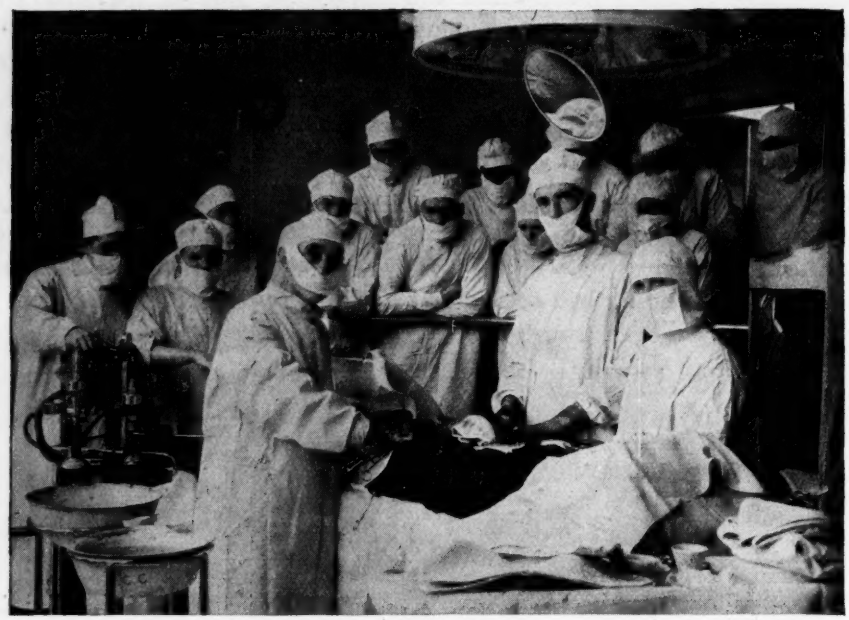

FIG. 407.—An operating theatre.

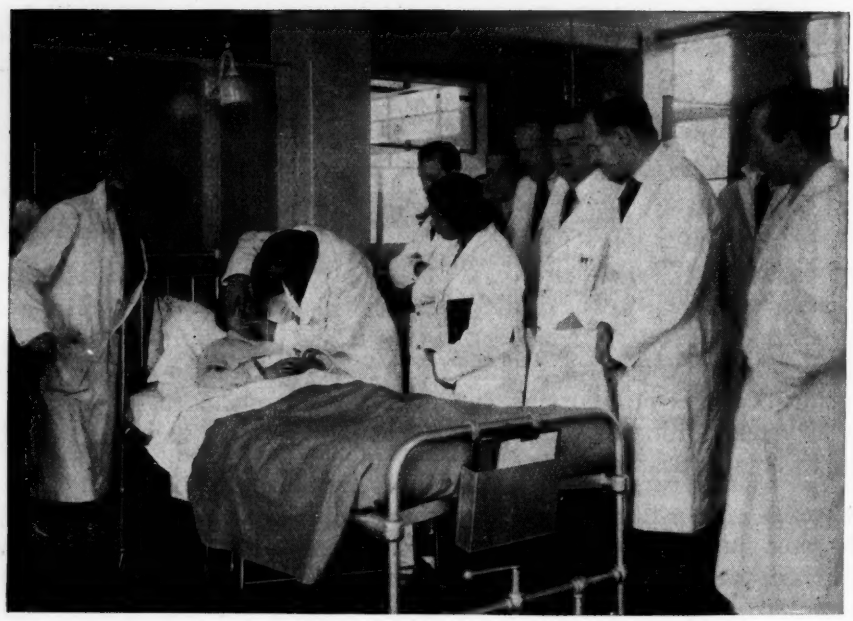

FIG. 408.—A hospital ward.

letting out purulent bile, but no stones. After this, whenever the sinus closed, there was recurrence of pain and jaundice. The heart showed senile myocardial degeneration with some fibrillation. Lipiodol injection into the sinus showed dilatation of the common bile-duct. The abdomen was opened by Kocher's incision. There were many adhesions between the gall-bladder, anterior abdominal wall, large intestine, and stomach. These were cut through and the gall-bladder and common duct fully explored. The common duct was very distended and contained a gall-stone. It was opened and the stone removed; a sound passed easily into the duodenum. The incision in the common duct was closed and a cholecystectomy performed. The abdomen was closed round a rubber tube draining the region of the common duct. The liver looked as if it were the seat of fatty degeneration. The stone removed was oval in shape and about $1\frac{1}{4} \times \frac{3}{4}$ in.

Case 3. A man of 36 with Pyloric Stenosis: Gastro-enterostomy and Gastrostomy.—Epigastric pain and tenderness for fifteen years. Vomiting began nine months ago and had increased in frequency and in amount. Marked loss of weight. Visible peristalsis from left to right over the umbilical region. X rays had shown great dilatation of a hypotonic stomach. After twenty-four hours the stomach still contained a large residue. Before the operation the stomach had been washed out daily for four days. One pint of intravenous glucose solution was given, and the abdomen opened by a midline supra-umbilical incision. The stomach was found to be greatly dilated and there was a cicatrizing ulcer in the first part of the duodenum. A posterior gastro-enterostomy and a Kader-Senn gastrostomy were performed. The abdomen was closed round the gastrostomy tube.

The stomach was not only very large, but was apparently atonic and certainly thin-walled, and it was on this account that the gastrostomy was combined with the other operation.

In the intervals between the operations we watched Mr. Feggetter doing a cystoscopy and a diathermy resection of the prostate. Also we had an interesting demonstration by Mr. Watson on some fracture cases, chiefly those of intracapsular fractures of the hip, which had been treated by nailing with the Smith-Petersen nail.

In the afternoon we made a tour of the medical school. Professor Kettle showed us the very well-equipped Department of Pathology. The post-mortem room is so large and well lit as to make a good demonstration and lecture theatre. There are tables for conducting four autopsies at the same time. In addition to the daily post-mortems there is a weekly lecture on selected autopsies performed during the week. The Museum is naturally small at the present moment, but it is beautifully arranged, and the descriptive catalogue gives clinical details, while stained microscopical sections are readily available for study. Biochemistry, bacteriology, and animal experimental work are amply provided for.

Dr. Duncan White showed us round the Department of Radiology. The diagnostic radiography is done in five separate rooms, which are used for general work, and for urological, dental, thoracic, and gastro-intestinal examinations respectively. There are twenty-four viewing boxes, and also all noteworthy or exceptional films are at once reduced to lantern slides, so that at a moment's notice there is always at hand a large number of typical or extraordinary pictures for lectures or demonstrations. There is also an X-ray therapy equipment, to which is to be added a radium department.

Later in the afternoon we were treated to a most fascinating demonstration by Professor Grey Turner on various products of his 'bow and spear'. We followed a short résumé of his pioneer work on cancer of the œsophagus, illustrated by specimens from different cases—we saw those specimens of cancer of the stomach, colon, and rectum which have justified him in regarding malignant disease as 'curable'.

Next morning we had various papers and demonstrations: Mr. Henry explained and carried out an extra-peritoneal method for the radical cure of inguinal hernia; Mr. Elliott Smith (a voluntary assistant), the operation of epididymis ligature for enlarged prostate; Mr. Feggetter gave a paper on diathermic resection of the prostate, which greatly impressed us with the potential dangers of the method. Mr. Watson demonstrated specimens from a case of fat-embolism, and we hope to publish this case at an early date. Dr. Aitken (Reader in Medicine) showed us the new type of flexible gastroscope, and we were able to see a gastro-jejunal ulcer on a gastro-enterostomy stoma quite clearly.

On leaving the British Post-Graduate School our feelings of enthusiasm for this great advance in medical education were mingled with those of envy—envy of the staff who have the honour of initiating this great venture, and envy of the post-graduate students who have facilities for seeing and learning those wonders of modern medical science which were not dreamed of when we were young.

So far as it is " the first step which counts ", the British Post-Graduate Medical School has already achieved a notable success, and is assured of a brilliant future.

EXPERIMENTAL SURGERY

TOXIN FORMATION IN BURNED TISSUES

By W. C. WILSON,* J. S. JEFFREY,* A. N. ROXBURGH,† AND C. P. STEWART*

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EXTENSIVE burning injuries in man give rise to severe systemic disturbances which change in nature, and evidently also in causation, as time elapses after the infliction of the injury; thus the clinical course of an extensive burn may be divided into several more or less distinct stages. A convenient division is into five stages: (1) Initial shock; (2) Secondary shock; (3) Acute toxæmia; (4) Septic toxæmia; and (5) Healing. The clinical features of each stage have been described and discussed in previous publications.[10, 11] We are concerned here only with the third stage of 'acute toxæmia', which begins between 6 and 50 hours after injury, which bears a close resemblance to other 'toxic' conditions, and which not infrequently ends in death at about 70 hours from the time of the burn.

The etiology of acute toxæmia has not yet been definitely established. Clinical and pathological studies[11] strongly suggest that it may be caused by the formation of a toxin or toxins in burned tissues and the absorption of such toxin into the circulation; some evidence from animal experiment can also be quoted in support of this view, which has met with general acceptance for many years. Recently, however, the view has been criticized adversely and on grounds which are vital for its acceptance—namely, the experimental evidence for the formation of toxins in burned tissue. It seemed to us that a decision on the question, which has important practical applications, was desirable and that it could be reached only by further investigation. The results of such an investigation are recorded in this paper.

REVIEW OF PREVIOUS EXPERIMENTAL INVESTIGATIONS

A very considerable bulk of experimental investigation on the cause of death after burns has accumulated during the past century. From this we have selected for review a few of the more complete and significant researches of comparatively recent date and relating particularly to the question under consideration.

Avdakoff, in 1876, was the first to report that the blood of a burned animal was toxic to healthy animals. Pfeiffer[3] in a convincing series of experiments showed that the urine and blood-serum of burned rabbits became toxic to mice, guinea-pigs, and also to rabbits. The toxic principles had both neurotoxic and necrotoxic actions; a study of their chemical characteristics, however, revealed little more than that they were labile substances with some properties in common with nucleoproteins and

* In receipt of a part-time grant from the Medical Research Council.

† Crighton Research Scholar.

snake venom. The toxic nature of the blood of burned animals was shown also by Robertson and Boyd.[4] In addition, they tested material from human cases of burns by its action on guinea-pigs; whole blood and cerebrospinal fluid proved poisonous, while blood-serum and blister fluid were innocuous.

Numerous experiments have been reported which seemed to implicate the burned area as the source of the toxic principles which appeared in the blood. By excision and transplantation of burned skin, Vogt[9] was able to prevent toxæmia in a burned animal and to produce it in the host. Confirmatory evidence was obtained from parabiosis experiments in which the unburned animal developed toxæmia unless separation was carried out within twelve hours. Robertson and Boyd[4] investigated the action of extracts of burned skin from human cases and animals; such extracts were lethal to guinea-pigs. Their conclusions were that the toxic substance in burned skin consisted of two portions, one of which was thermostabile, diffusible, and neurotoxic, the other thermolabile, colloidal, and necrotoxic; chemically it was composed of primary and secondary proteoses.

Although there was a large measure of agreement that toxic substances were elaborated in burned tissue and absorbed into the blood-stream, opinion regarding the nature of the toxins was far from unanimous, and many improbable and even bizarre suggestions were made, frequently without basis of evidence. Little information of value has been gained from attempts at chemical identification and isolation; some facts, however, have been recorded which might be held to point to products of protein cleavage.

The theory of toxin formation and absorption has not, however, been universally accepted. Underhill and his associates, for example, attributed the systemic disturbances of extensive burns mainly to increased concentration of the blood;[6, 7] they also held that absorption of a toxin from burned tissues was highly improbable. Direct evidence against the toxin theory was claimed by Underhill and Kapsinow,[8] who repeated the experiments, and failed to confirm the results, of Robertson and Boyd. They could discover no significant difference in action between extracts of burned skin and extracts of normal skin, nor between blood from burned animals and blood from normal animals.

Consideration of the above and other literature has led us to the conclusion that the evidence from animal experiment for a specific 'burn toxin' is inconclusive, though there is much that is suggestive. Proof of the toxin theory would require the demonstration and identification of the toxic substances not only in the burned area but also in the circulating blood.

We decided to investigate first the question of toxin formation in burned tissues.

METHODS

The experiments were carried out on rabbits. When excessive heat is applied to the skin of a rabbit there is no blistering, but a considerable quantity of œdema fluid accumulates in the subcutaneous tissues; swelling is obvious at 1 hour and increases up to 24 or 36 hours, after which a slow absorption begins. Very mild degrees of injury by heat are followed by hyperæmia and slight œdema, which disappear in a few days. After exposure to higher temperatures œdema is more conspicuous and lasts for a week or longer; the skin, however, finally regains its normal condition except for a persistent scaliness and sometimes a few small isolated

areas of necrosis. Following still more severe injury, the affected skin dies and is finally thrown off as a dry, wrinkled, black slough; eventually such areas are invaded by micro-organisms. Our object was to produce by the application of heat an accumulation of œdema fluid without necrosis of skin and without injury to internal organs by the direct action of heat.

In rabbits the skin of the abdomen was shaved and cleansed by ether and alcohol over an area comprising about one-sixth of the total skin surface. Under full ether anæsthesia, and with aseptic precautions, an incision was made in the middle line and the skin of the area together with subjacent platysma muscle dissected up as two lateral flaps. Heat was then applied to the deep surface of the flaps, except over a thin strip on either side of the incision, by a very brief exposure to the diathermy current; the active electrode was a flat disc about 1 cm. in diameter. The incision was closed and in most instances the burned area was protected by collodion applied over gauze. In other experiments the injury was effected by a jet of steam applied to the external skin surface; by either method an injury of the required severity was obtained. The animals subsequently showed no special untoward symptoms.

At varying intervals after burning, the œdema fluid which had accumulated was collected in the following way. The animal was anæsthetized, the area cleansed with ether, the skin and œdematous subcutaneous tissue dissected off, leaving behind a vertical strip on either side of the operative incision, and as much free œdema fluid as possible was collected. Prior to the dissection a small incision was made through sterilized skin and a few drops of œdema fluid were taken for aerobic and anaerobic cultures. When the burned area had been excised the animal was killed. The excised tissue was cut into small pieces and the œdema fluid expressed from it through a Büchner filter with suction attached. Throughout the procedure all precautions were taken against bacterial contamination of fluid and tissue. It was possible to obtain by this method from one rabbit 10 to 20 c.c. of œdema fluid which, on account of hæmoglobin content, was straw-coloured or pink and which contained also small particles of tissue. The solid particles could then be removed by centrifugation.

The toxicity of œdema fluid was tested by intravenous injection (into an ear vein), by subcutaneous and intraperitoneal injection in unanæsthetized rabbits, and by intraperitoneal injection in mice.

RESULTS

Summary of Results of Intravenous Injection of Œdema Fluid in Healthy Rabbits.—*Table I* shows the results of injecting œdema fluid removed at different intervals from burned areas.

The quantities injected were from 2 to 20 c.c.; 5 to 10 c.c. was an average dose. It will be seen that after a certain period following burning the œdema fluid became highly toxic, and that the development of toxicity was gradual. At forty-eight hours the fluid was frequently lethal.

The Rôle of Bacteria.—In earlier experiments cultures were made from the fluid after removal of tissue and extraction of fluid; in these circumstances a profuse growth of organisms was obtained in both aerobic and anaerobic cultures. When, however, the method of taking cultures through a small incision was employed the cultures proved almost always negative. This was the case also when injury was produced by steam. It was clear that as a rule no organisms were present in the œdema fluid before removal of the burned tissue, and that any organisms in the

fluid used for injection had been introduced during the procedure of extracting the œdema fluid. Moreover, a final survey showed no significant difference in toxicity of œdema fluid whatever the results of cultures. We have, therefore, reported in *Table I* experiments irrespective of the results of bacteriological cultures.

Table I.—Results of Intravenous Injections of Œdema Fluid

Injections	Total Animals	Died within 10 Days	Died after 10 Days	Ill but Survived	Not Obviously Affected
A. Œdema fluid removed at :—					
4 hours	6	0	0	0	6
6 hours	2	1	1	–	–
12 hours	2	2	–	–	–
24 hours	6	2	–	2	2
27 hours	2	1	1	–	–
48 hours	44	38	2	3	1
B. Œdema fluid removed at 48 hours and passed through Seitz filter	15	7	–	–	8

When the fluid was passed through a Seitz filter to remove organisms, there was usually a diminution in toxicity; toxicity, however, was not abolished (*Table I*). Diminution in toxicity occurred irrespective of the results of cultures made previous to filtering, and was apparently not due merely to removal of organisms.

We have concluded that the development of toxicity of œdema fluid was independent of the growth of organisms in the burned area.

Control Experiments.—Œdema fluid removed at four hours after injury was invariably innocuous even in large doses. These experiments served as a control series. In addition, control experiments were done by injecting saline emulsions of large areas of skin immediately after burning, and saline emulsions of normal skin. In no case was any toxic action observed. In both control series injections were made, without deleterious effect, when previous centrifuging of the samples had been omitted. Thus it was shown that the toxic effects of œdema fluid were not merely a result of embolism by tiny particles of tissue. A further possibility had to be eliminated. Roome and Wilson[5] found that œdema fluid from the traumatized muscles of a limb, on injection into healthy animals, caused immediate death by extensive intravascular clotting. We have not observed intravascular clotting after injecting œdema fluid from burned areas.

Properties of Œdema Fluid Removed at Forty-eight Hours after Burning.—It was found that at forty-eight hours the amount of fluid present was nearly maximal and that usually a high degree of toxicity had developed. After this time the risk of bacterial invasion of the œdema fluid became steadily greater. The properties of fluid removed at forty-eight hours were therefore investigated.

Effects of Injection.—Samples of fluid differed in toxicity, and there was also a considerable individual variation in susceptibility of animals injected. Sometimes death occurred within a few seconds after 3 to 5 c.c. had been injected; in such cases generalized convulsions supervened, respiration failed, and the heart stopped. In other instances the animal, after several minutes, developed signs of muscular

weakness, laboured respiratory movements, increased heart-rate, pallor of the ears, and dilatation of pupils; sometimes urine and fæces were voided. The weakness became gradually more marked, the rectal temperature fell after a

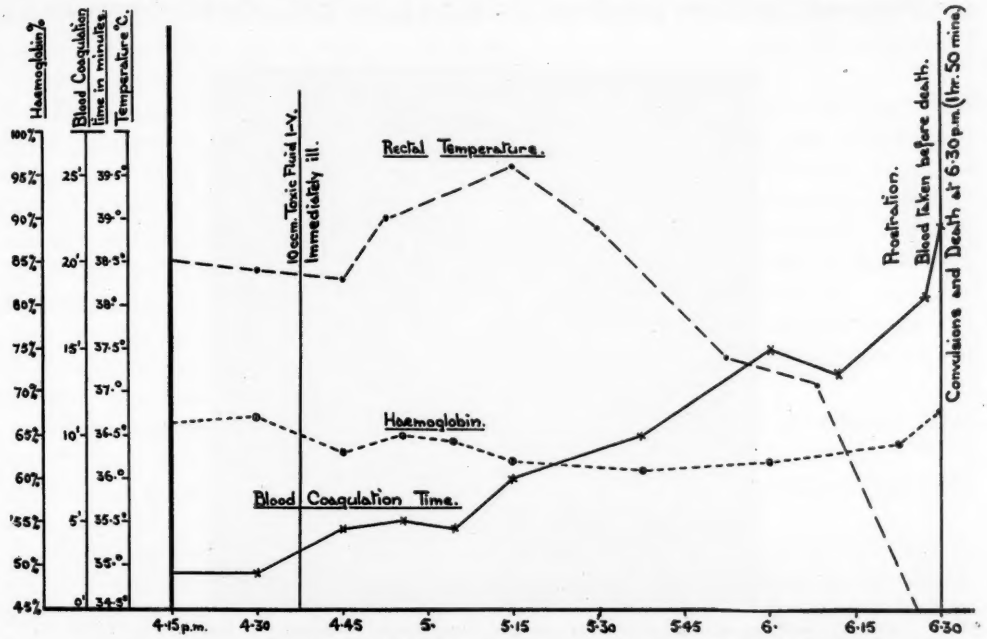

FIG. 409.—Chart showing changes in blood concentration, rectal temperature, and blood coagulation time after intravenous injection of toxic fluid.

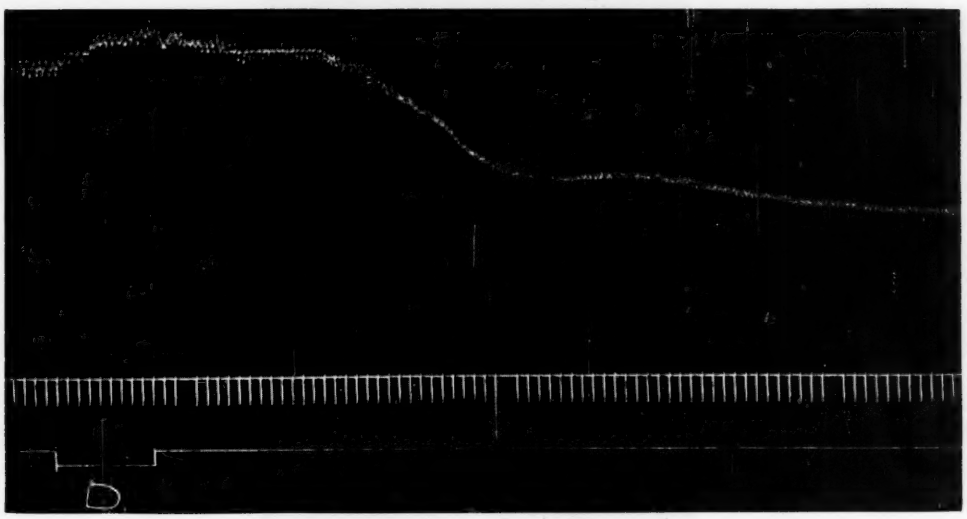

FIG. 410.—Effect of intravenous injection of toxic fluid on the blood-pressure. At D, Injection of 1 c.c. of fluid. Fall of pressure from 110 mm. Hg to 56 mm. Hg in 2 minutes. Time marker, 2 sec.

preliminary rise, and death occurred from one to five hours after injection, sometimes preceded by muscular twitchings or generalized convulsions. Blood withdrawn from an ear vein showed no alteration in hæmoglobin content; the

blood coagulation time was usually increased. *Fig.* 409 illustrates the changes in a typical experiment.

Samples of fluid which caused death within a few hours were tested by injection in rabbits under urethane anæsthesia (1·0 to 1·5 g. per kilo); the blood-pressure was

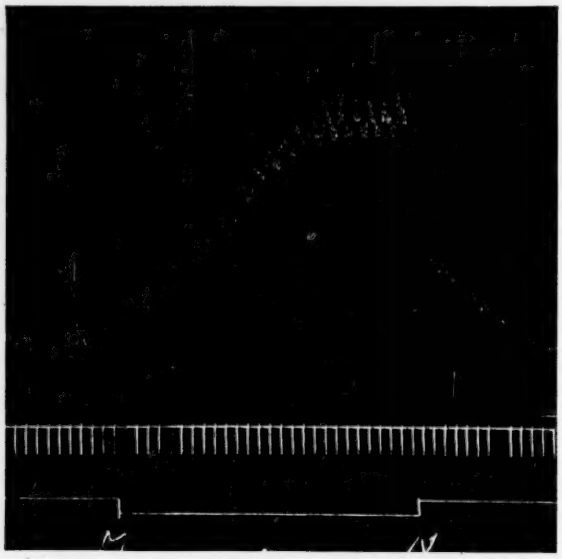

FIG. 411.—Effect of clamping aorta on the hypotension produced by toxic fluid. M to N, Descending aorta clamped. Blood-pressure rose from 30 mm. Hg to 112 mm. Hg. Time marker, 2 sec.

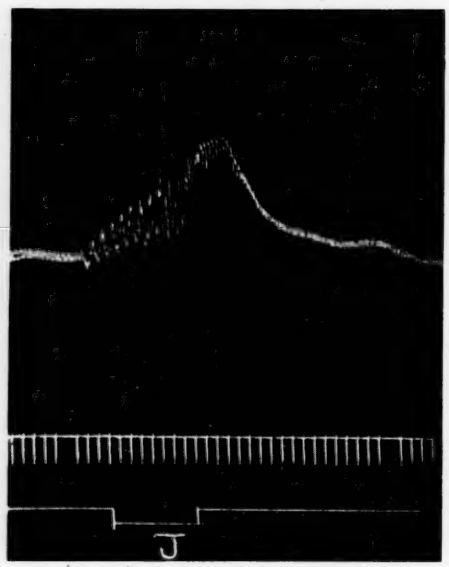

FIG. 412.—Effect of asphyxia on the hypotension produced by toxic fluid. At J, Obstruction of trachea. Blood-pressure rose from 64 mm. Hg to 104 mm. Hg. Time marker, 2 sec.

recorded and a tube tied in the trachea. The blood-pressure sometimes fell immediately after injection and the fall was permanent (*Fig.* 410); in other instances the fall was very slow but progressive. During the collapse of arterial pressure the

thorax was opened under artificial respiration; the heart was beating vigorously though rapidly, and the great veins and the right heart were not dilated. Clamping the thoracic aorta for a short period produced a rise in blood-pressure to the original level (*Fig.* 411). Clearly the hypotension was not due to a primary failure of heart action. Nor was it due to exhaustion of the vasomotor centre, since this responded to temporary asphyxia by a distinct rise in blood-pressure (*Fig.* 412).

In the majority of experiments death occurred at much longer intervals than those mentioned above. The average period of survival of animals which died within ten days, excluding those which succumbed immediately, was calculated as thirty-nine hours. This figure is probably higher than the true period, since in a number of instances death had occurred some hours before it was noted. Usually no change was evident for some hours after injection; in one, two, or more days the animal began to refuse food, became gradually prostrated, and died. In a few cases the animal was immediately ill and remained so for a number of hours but ultimately recovered; in others, and these were numerous, a period of apparent recovery was followed by relapse and death after several days.

Subcutaneous and Intraperitoneal Injection in Rabbits.—Œdema fluid proved toxic when injected subcutaneously or intraperitoneally in rabbits; for a lethal effect quantities larger than those which were used in intravenous injection were required.

Intraperitoneal Injection in Mice.—The quantities injected were 0·5 to 1·0 c.c. The results were closely parallel with those of the same samples in rabbits. The action of the fluid was sometimes striking; death occurred at twenty-four to forty-eight hours, and was preceded by prolonged spasm of certain muscle groups, so that the head was flexed, the back arched, and the limbs and tail extended. Control inoculations of blood and serum from normal rabbits were negative.

Post-mortem Changes.—In rabbits which succumbed immediately no special changes were observed; there was no evidence of bronchial constriction, and, as already stated, intravascular clotting was not found. In animals surviving for a few hours or longer the liver was the organ which sustained particular damage; abnormalities in the kidneys, gastro-intestinal tract, brain, heart, lungs, or suprarenals were slight or absent. The liver in a typical case was pale, yellowish, sometimes of a 'nutmeg' appearance, and abnormally firm in consistence. Microscopic section showed fatty degeneration, which was confirmed by appropriate staining, involving mainly the cells of the centres of the lobules (*Figs.* 413, 414). The liver damage was most severe in animals which died during the first few days; if the survival period were more than a week the changes were less constant and less conspicuous.

The Nature of the Toxins.—The gradual development of toxicity of œdema fluid in the absence of bacterial growth strongly suggested that autolysis of injured tissue was responsible for toxin formation. The possibility that the toxic principles were products of protein cleavage was therefore tested. Various protein fractions were made and injected into rabbits; the results are tabulated in *Table II.*

The albumin fraction was separated from the globulin by appropriate saturation with ammonium (or magnesium) sulphate and the sulphate ions were removed by dialysis through a collodion membrane. The globulin fraction separated out during dialysis into euglobulin and soluble pseudoglobulin fractions. In some cases the two globulin fractions were injected separately. The results showed that the

toxic principles were present in both albumin and total globulin fractions, but were probably associated mainly with the globulin fraction. Alteration of the *p*H so as to secure more complete precipitation of globulin did not affect the toxicity of the albumin fraction, and there was no significant difference in toxicity of the

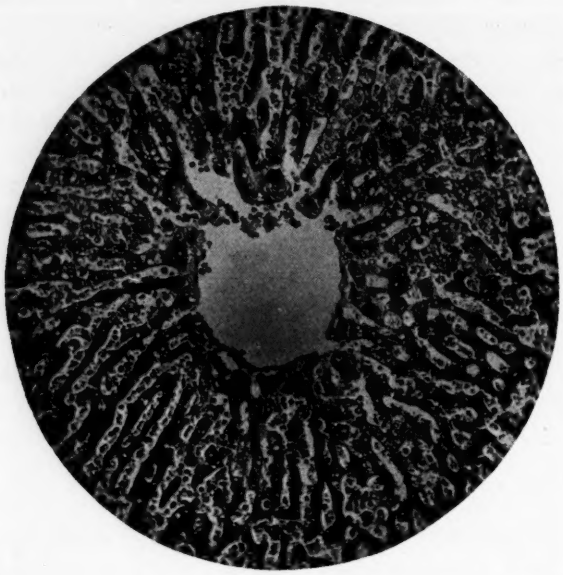

FIG. 413.—Fatty degeneration of liver-cells. Animal died six days after injection of toxic fluid.

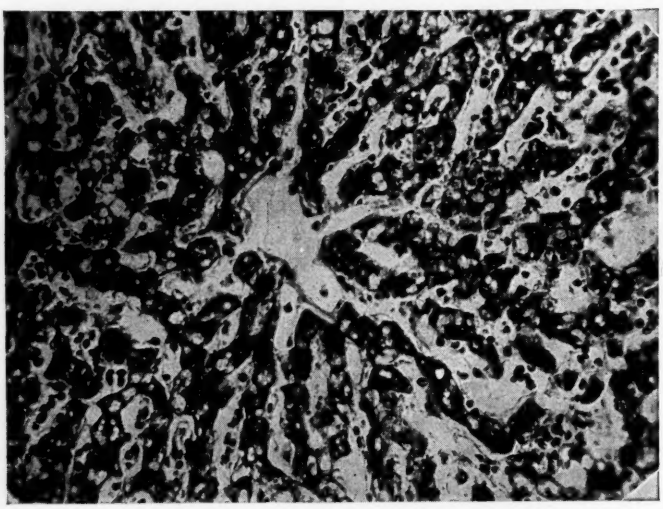

FIG. 414.—Fatty degeneration of liver-cells. Animal died 17 hours after injection.

two globulin fractions. The mother liquor after removal of protein fractions was non-toxic.

Previously it had been shown[2] that trichloracetic acid extracts of burned skin, made by the method of Chang and Gaddum,[1] contained an unidentified depressor

substance; extracts of normal skin of rabbits, however, contained the depressor substance in equal or greater amounts. In the present investigation trichloracetic acid extracts of œdema fluid were injected into unanæsthetized rabbits in quantities corresponding to several lethal doses of crude fluid. In one case, where a large dose was injected, death occurred. However, the extract of normal skin and an equal quantity of fluid from a blank extraction also proved fatal. It was therefore impossible to attribute death following administration of trichloracetic acid extract of œdema fluid to any constituent of the fluid resulting from burning. The negative results of the remaining injections suggested that the toxic principles of œdema fluid were not present in such extracts.

Boiling œdema fluid or heating it at 60° C. for 30 minutes lessened the toxicity (*Table II*).

Table II.—Results of Injections of Chemical Fractions

Injections	Total Animals	Died within 10 Days	Died after 10 Days	Ill but Survived	Not Obviously Affected
Albumin fraction ..	9	5	1	–	3
Total globulin fraction ..	12	9	2	–	1
Euglobulin fraction ..	3	1	–	–	2
Pseudoglobulin fraction ..	4	1	–	–	3
Mother liquor	3	–	–	–	3
Trichloracetic acid extracts	5	1	–	–	4
Toxic fluid after boiling ..	4	1	1	–	2
Toxic fluid after heating at 60° C. for 30 min. ..	2	1	–	–	1
Toxic globulin fraction after boiling	3	1	–	1	1
Toxic albumin fraction after boiling	1	–	–	–	1

The Rôle of Enzyme Action.—In order to test whether toxicity could be produced in œdema fluid when organisms were excluded we carried out the following experiments. Œdema fluid was filtered through a Seitz filter and kept in a refrigerator at 4° C. The fluid, originally non-toxic, proved lethal 10 days later, but became innocuous again at 100 days. Similarly, filtered non-toxic fluid when incubated at 37° C. was strongly toxic at 10 days but had lost toxicity by 30 days. The results suggested that toxin production was by enzyme action. Bacterial growth would presumably have led to a sustained level of toxicity.

Attempts to immunize rabbits by repeated injection of small quantities of toxic fluid proved unsuccessful.

DISCUSSION

In these experiments it has been demonstrated that œdema fluid extracted from a burned area is toxic to healthy animals of the same species. By injecting œdema fluid itself, we avoided fallacies connected with methods of chemical extraction; other fallacies such as the effects of embolism or of introducing hæmocoagulants into

the circulation were also ruled out. The development of toxicity was slow, and, although the experiments were not sufficiently numerous to indicate it clearly, probably steady and progressive, up to forty-eight hours at least. Fluid at four hours after injury was not toxic. Clearly the toxic principles were not formed immediately by the action of heat. We were able to exclude with reasonable certainty the action of micro-organisms and to furnish also some direct evidence that toxic substances could be formed in œdema fluid by enzyme action after organisms had been removed by filtering. In view of these facts it seemed justifiable to conclude that autolysis of injured tissue was the mechanism of toxin elaboration.

The actions of œdema fluid indicated that more than one toxic substance was present. Some samples had a rapidly lethal, apparently neurotoxic, action which was entirely absent from others. Samples which caused death within a few hours did so by virtue of some component which had many points of community with histamine and other capillary poisons. Histamine itself, however, was not then the main toxic element, since, among other reasons, the fluid caused a marked fall in blood-pressure in the anæsthetized rabbit. When, as in the majority of experiments, œdema fluid caused death after a considerable number of hours, there was sometimes little to indicate its mode of action; the chief, though not invariable, effect was to produce degenerative changes in the liver cells. The action of the toxic principles requires further investigation.

In respect of identification of the toxins, the study is also incomplete. The toxic bodies were associated mainly with the globulin fraction, though the albumin fraction was only slightly less implicated, and thus were brought into the category of protein derivatives. The evidence given by the action of trichloracetic acid extracts, though complicated by the results of control experiments, may be considered as an indication that the more potent of the poisons were derivatives of a higher order than amino-acids or lower peptides. Even if this evidence be disregarded, that from the injection of mother liquor and from the effect of boiling or heating œdema fluid still points to the same conclusion.

The burned animals were in most instances very little affected by the injury during the period of survival allowed, i.e., forty-eight hours—a feature which might be regarded as an objection to the thesis of toxin formation and absorption. In our opinion, however, the objection is not serious. Since the burned area was of limited extent, and the development of toxicity was gradual, the quantity of toxin absorbed during the period of survival would, it is reasonable to expect, be small. Moreover, the capacity of animals to survive repeated small doses of toxin was proved in experiments on attempted immunization. We do not intend to discuss here the question, raised by Underhill and others, of the rate of absorption from burned areas; this feature has not been investigated, and, furthermore, its importance in relation to the toxin theory must rest principally upon the conditions of absorption in human beings.

A comparison of the toxic effects in animals of injected œdema fluid with the clinical condition of acute toxæmia is of special interest. In many respects there is a marked similarity; the resemblance is perhaps strongest in the post-mortem changes, because the striking and constant feature of fatal toxæmia is necrosis and fatty degeneration of the liver-cells in the centres of the lobules. Even without these points of similarity the demonstration of toxin formation in burned areas is in itself suggestive that the action of circulating toxins may play an important part

in the systemic disturbances of burns. Taken in conjunction with evidence, which will be detailed in a separate paper, that, in human cases, increased concentration of the blood, early bacterial infection, and changes in blood chemistry are not essential causes of toxæmia, the results of this investigation, although in some respects incomplete and as yet unaccompanied by a demonstration of toxic properties in the blood of burned animals, might seem of particular significance.

CONCLUSIONS

1. Œdema fluid which accumulates in a burned area gradually acquires toxic properties; when collected at forty-eight hours after burning it is frequently lethal to healthy animals of the same species.

2. The development of toxicity is independent of the action of bacteria; there is evidence that autolysis of injured tissue is the responsible mechanism.

3. A study of the actions of toxic œdema fluid suggests the presence of more than one toxic component, and that the proportions of the components vary in different samples.

4. The effects produced include toxic action on the nervous system, circulatory depression, and degeneration of liver-cells.

5. The toxic principles are associated mainly with the globulin fraction. The evidence at present available indicates that the more active principles are higher protein derivatives.

6. The relation of these results to the etiology of acute toxæmia of burns in human cases is mentioned. Features which require further investigation are indicated.

We are indebted to Dr. C. P. Beattie, Bacteriology Department, University of Edinburgh, for assistance and advice in the bacteriological part of the work. The expenses were in part defrayed by grants from the Earl of Moray Fund, University of Edinburgh.

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SHORT NOTES OF RARE OR OBSCURE CASES

ILEOCOLIC INTUSSUSCEPTION OF INFANCY TENDING TO NATURAL CURE

By JAMES COOK, Birkenhead

The following case of ileocolic intussusception is of interest, presenting as it does some unusual clinical features. It also illustrates the manner in which the critical period may be tided over pending complete separation of the intussusceptum in cases of 'natural cure'. That a relatively formidable operation was well tolerated by one of such tender years was due to the relief secured from obstruction by the formation of a fistula between the intussusceptum and intussuscipiens prior to the operation.

History.—A. L., a female aged 13 months, was admitted to Birkenhead Municipal Hospital on June 15, 1936, having had attacks of vomiting daily for six days. On June 12, following a constipated motion some blood 'oozed' from the anus; thereafter the bowels remained confined until June 14, when a sharp attack of diarrhœa occurred, the stools being dark in colour. There had been several attacks of vomiting on the morning of admission and the bowels had not then moved for twenty-four hours. The infant was a first child, had been bottle fed, and had had no previous illnesses.

On Examination.—The infant was well nourished, was somewhat fretful, and had flushed cheeks. The teeth were good, and no abnormalities were found in the heart or lungs. The abdomen was distended but there was no palpable mass. Rectal examination was negative. Temperature was 98° F., pulse 110, and respiration 22. A soap-and-water enema resulted in a dark, fluid offensive motion. There was considerable mucus present and much flatus was passed.

June 16.—Abdominal distension was much less marked. No mass was palpable. Bowels did not open. There was no more blood noted on rectal examination. Vomiting occurred thrice during the night. T. 100°; P. 120; R. 28.

June 17.—A little greenish fluid was vomited. A normally coloured motion was passed. T. 97°; P. 118; R. 26.

June 18.—There was no more vomiting. Spasms of abdominal pain occurred. The abdomen remained distended. A normal motion was passed in the morning. T. 97·4°; P. 120; R. 26.

June 19.—Abdominal distension was less marked. A formed motion was passed. The examination for occult blood was positive. There was no further vomiting. T. 100°; P. 120; R. 28.

June 20.—Abdomen still distended. Stools of normal consistence passed. Occult blood positive. T. 98°; P. 124; R. 28.

June 21.—There was no vomiting, rigidity, or apparent tenderness. Abdominal distension persisted. Rectal examination was negative. Bowels were open thrice, the motions being relaxed and offensive. T. 98·8°; P. 118; R. 28.

June 22.—Abdominal distension more marked and visible peristalsis noted for first time. Vomiting recurred. There was offensive diarrhœa, and blood and mucus were passed per rectum. T. 103·8°; P. 150; R. 34.

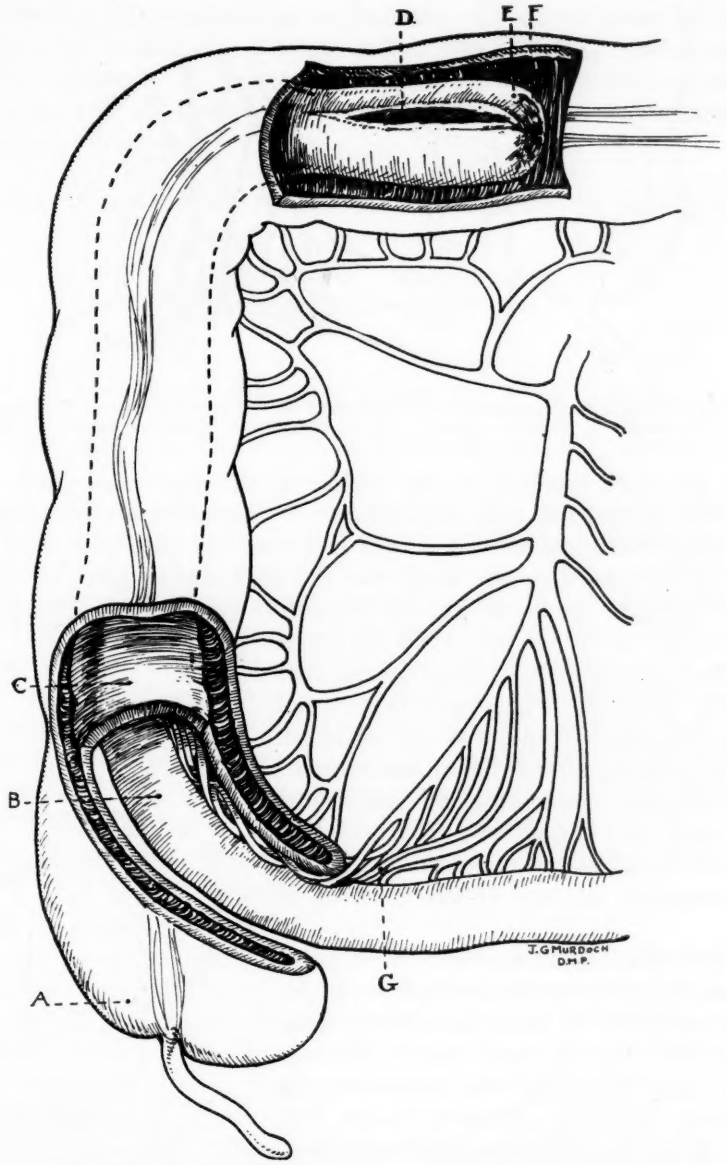


FIG. 415.—Diagrammatic representation of appearances found on opening abdomen. A, Cæcum with appendix; B, Entering layer of intussusceptum; C, Returning layer of intussusceptum; D, Fistula between ileum and transverse colon; E, Apex of intussusception; F, Lumen of ileum occluded by inflammatory œdema; G, Indrawn mesentery.

This was the 13th day of the child's illness, yet her general condition remained fairly satisfactory. Emaciation was absent. There had been no vomiting from June 16 to June 22, and formed motions were passed on the 8th, 9th, and 10th

days of the illness. The recurrence of vomiting together with the appearance of visible peristalsis on June 22 called for abdominal section.

OPERATION.—Under chloroform and ether anæsthesia the abdomen was opened through a right paramedian incision and an intussusception of the ileocolic type

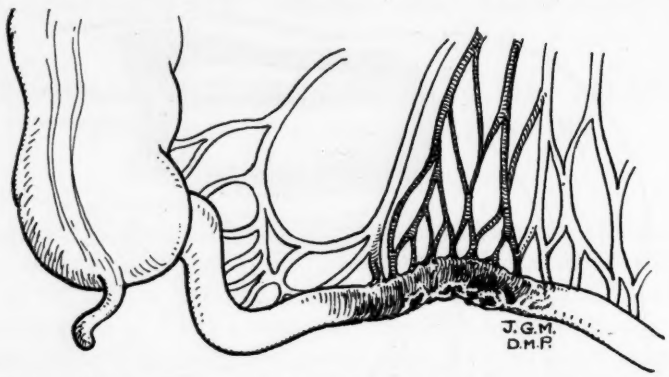


FIG. 416.—Illustrates the appearances after reduction of the intussusception. The gangrenous part of the ileum, its perforated portion, and the thrombosed mesenteric vessels are shaded.

was found, the apex reaching to the middle of the transverse colon (*Fig.* 415). Reduction was carried out with especial care. There was a considerable amount of plastic lymph between the two serous layers of the intussusception, and when reduction was completed it was found that the apex had sloughed so that a fistula had existed between the ileum and the transverse colon. The mesenteric vessels in relation to the necrotic portion of the ileum were thrombosed (*Fig.* 416). The intussuscipiens was relatively healthy. The appendix was not involved in the process and was not removed. The affected portion of the ileum was resected and an end-to-side anastomosis of ileum into ascending colon was carried out (*Fig.* 417). A rubber tissue drain was passed down to the site of the anastomosis and the abdomen closed in layers.

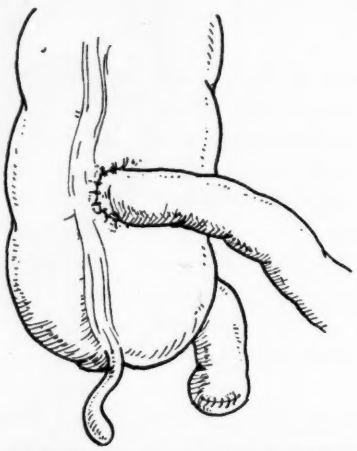


FIG. 417.—Represents appearance after resection and end-to-side anastomosis.

POST-OPERATIVE COURSE.—On the evening of operation the temperature reached 103·3° F., remained elevated for six days, and then returned to normal. The bowels were opened three to four times daily following the operation, the motions being somewhat offensive for the first two days. At no time did the child's condition after the operation cause concern. There was no recurrence of vomiting, sterile water and later glucose water and Benger's food being well tolerated.

COMMENT.—The case is one of ileocolic intussusception in a female infant. Most children suffering from this disease die within the first week if not operated upon, yet this child was admitted to hospital on the sixth day and survived a severe abdominal operation on the 13th day of her illness. It is estimated that approximately 2 per cent of cases not operated upon may survive, ' natural cure ' following

sloughing, separation, and expulsion of the intussusceptum. The sloughing probably commences at the apex of the intussusceptum, and it is most likely that in those cases surviving, nature first establishes (as in this case) drainage of the obstructed bowel. Indeed it would appear that in this case such drainage had been established prior to admission to hospital. Offensive stools in the absence of a palpable abdominal mass, together with a swinging temperature, gave rise to the erroneous diagnosis of primary ileocolitis. The infection actually present in this case arose in all probability from the gangrenous process present in the intussusceptum.

The author wishes to thank Dr. J. G. Murdoch for the accompanying illustrations.

A CASE OF MULTIPLE MYELOMA

By Lieut.-Colonel M. M. CRUICKSHANK, I.M.S., Madras, India

This case was one in which unilateral proptosis due to an intra-orbital tumour developed in the course of a year in association with X-ray appearances in the skull indicative of isolated areas of rarefaction. The X-ray appearances were not unlike those seen in skeletal lipoid granulomatosis, the Schüller-Christian syndrome. The patient was referred to the Ophthalmic Hospital, Madras, for opinion, where Col. R. E. Wright performed tarsorrhaphy, explored the orbit, received a pathological report of a round-celled sarcoma, and treated the intra-orbital growth with radium implantation. The mass disappeared after 864 mg.-hr. had been given, and, as it was considered that nothing further could be done in view of the fact that the condition was probably multiple myeloma, a diagnosis supported by a later histopathological report, the patient was returned to Madura. An X-ray print shows the condition of the skull. Eventually the whole skeleton showed similar invasion. The detailed notes of the case are as follows:—

G., Hindu, male, age 30.

May 11, 1934.—Seen in the Ophthalmic Department, Government Headquarter Hospital, Madura, complaining of pain in and protrusion of the left eye—duration six months. There was marked proptosis down and out of left eyeball, with definite uniform diffuse swelling, extending temporal-wards from the outer orbital margin, and inwards into orbit, causing proptosis of the eyeball (*Fig.* 418).

R.E.V., 6/6; L.E.V., 6/36.

Pupils equal and active. Fundi—right and left—normal.

All movements in both eyes free except upward in left.

Blood for Wassermann reaction, negative.

Urine: reaction, acid; albumin and sugar, nil; sp. gr., 1010.

July 12.—Patient again seen. Tumour mass had definitely increased in size and a diagnosis of ? sarcoma extending into orbit was made. X rays showed 'honeycombing', a number of small rounded areas of rarefaction, suggestive of the changes seen in the Schüller-Christian syndrome.

Patient sent to Col. Wright for opinion. Col. Wright removed part of the tumour for microscopic examination, the pathologist at first reporting lympho-sarcoma. Tarsorrhaphy of outer third was done, and radium implanted—total dose 864 mg.-hr.

Orbital growth disappeared, and proptosis became reduced to vanishing point.

Sept. 3.—Patient again seen. R.E.V., 6/6; L.E.V., 6/60.

I then lost sight of the patient, but Dr. Natesan, Radiologist, Madura, who had been asked to trace him, did so, took a fresh series of X-ray films, and sent

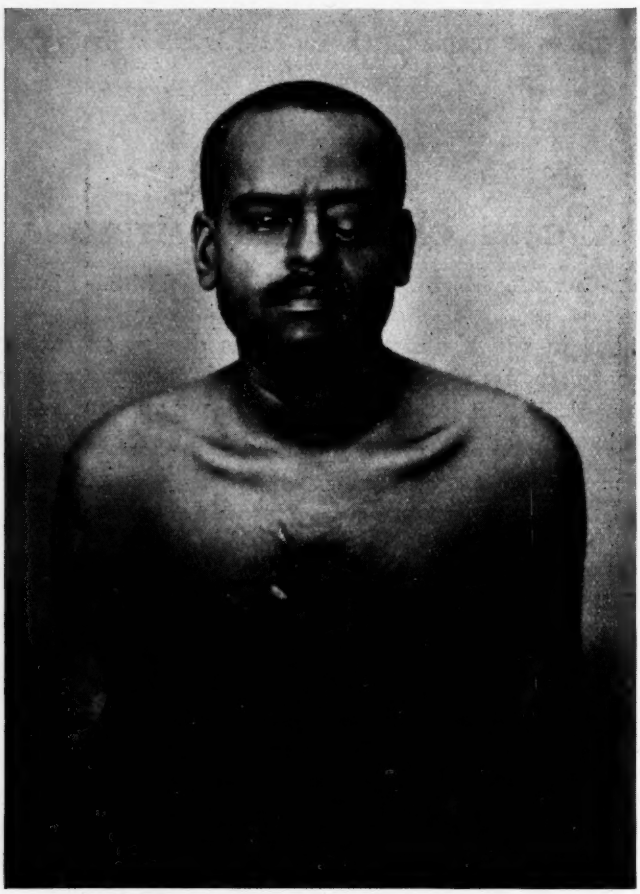


FIG. 418.—Showing proptosis and displacement of the left eyeball.

them to me at the General Hospital, Madras. The films showed an increase of rarefied areas all over the cranium (*Fig* 419).

Nov. 20.—Patient admitted to the General Hospital, Madras, for further investigation. He now complained of severe pain over the right side of chest and back, and the headache, though of less severity than formerly, still persisted. Proptosis of the left eyeball was again present. Patient stated that his general health had been good, though he looked thinner and less well nourished than he did in May, 1934.

Blood Chemistry (investigated by Dr. Mannadi Nayar).—

Blood-sugar	109·6 mg. per 100 c.c.
Blood-lipoid	270·8 „ „ „ „
Blood-cholesterol	181 „ „ „ „

Blood-count.—

R.B.C.	4,650,000
W.B.C.	9375
Polymorphs	66 per cent
Lymphocytes	30 „ „
Eosinophils	1 „ „
Mononuclears	3 „ „

Wassermann reaction, negative; Kahn test, negative.

FIG. 419.—Radiograph of skull.

Radiography.—X-ray examination of the skeleton was carried out by Dr. Pillai, of the Barnard Institute of Radiology. The radiological findings were :—

Chest : There is a tendency to early fibrotic changes in the right lung, chiefly at the upper lobe. Tendency to calcification of the costal cartilages. Hilar shadows rather dense.

Humerus : There is a large area of atrophic changes of the fibrocystic type in the upper half of the left humerus and a small area in the lower end on its external aspect. There are similar changes noted in the whole of the right humerus (*Fig.* 420).

Scapula : The left scapula shows similar changes at the lower half of the axillary border (*Fig.* 420).

Radius : The upper end of the left radius shows an area of atrophic changes at the level of the tubercle of the radius. The upper end of the right radius also shows similar changes of an early nature.

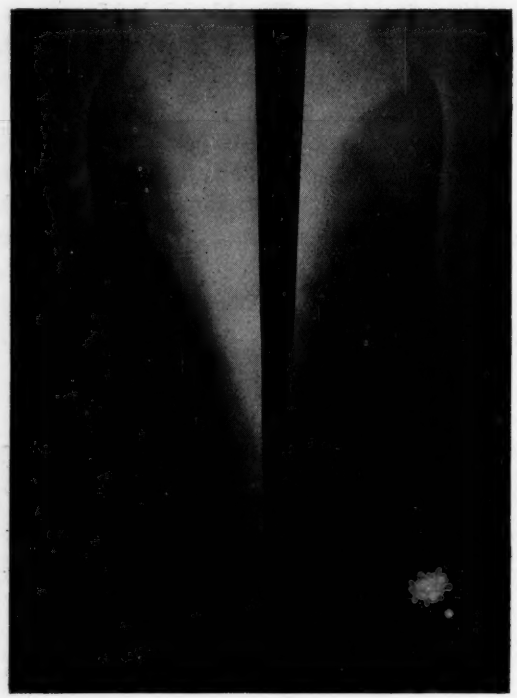


FIG. 420.—Radiograph of humeri and scapulæ.

FIG. 421.—Radiograph of pelvis and upper parts of the femora.

Lower limbs: The alæ of the iliac bones, the pubis and the rami, and the upper ends of the femora also show similar changes (*Fig.* 421).

Jan. 25, 1935.—Arrangements had been made to repeat all blood examinations but the patient suddenly left the hospital and proceeded to his home.

June 6.—Dr. Natesan, who had got in touch with the patient, sent the following note from Madura:—

General Condition.—Highly emaciated—"skin and bones"; bed-ridden; puffiness of the cheeks and feet; markedly anæmic; abdomen tympanitic.

Skull.—Left eyeball proptosis as before. A slight softish tumour mass, the size of a bean, has appeared just above the glabella. Probably on account of his emaciation the left parietal eminence appears slightly bigger than normal.

Eye.—Vision in left eye—perception of light; right eye—normal.

Respiratory System.—Slight cough. Wooden dullness over the whole of the chest. ? further deposits in lung.

Urine.—Scanty, high-coloured, loaded with albumin. Bence-Jones reaction—negative.

June 18.—Dr. Natesan reported that the patient died on June 15.

FINAL HISTOPATHOLOGICAL REPORT (Dr. A. N. Goyle).—Myeloma.

Detail of Cellular Structure.—"The cells themselves chiefly conform to two types, one fusiform or ovoid with deep-staining nuclei with indistinct chromatin, and the other with large rounded light-stained nuclei showing well-defined chromatin nodes. These cells show very little protoplasm. Under the oil-immersion lens, the nuclear chromatin is not arranged in a regular cartwheel fashion as in the plasma cell, but there is some resemblance in the nuclear structure to those cells. The protoplasm is scanty and can hardly be made out. It does not show the basophilic character of the plasma cell. Here and there, large cells with large nuclei can be made out. Mitotic figures are very few in the section. I think the dark-staining cells are more or less of the same type, where the nuclei have undergone compression. In some places the nucleolus has taken an acidophilic staining. With Unna Pappenheim, these cells do not show the differential staining of plasm cells, since the protoplasm can hardly be made out. On the whole the nuclear structure is more that of an endothelioid type of cell."

A CASE OF DIAPHRAGMATIC HERNIA

BY T. W. MIMPRISS

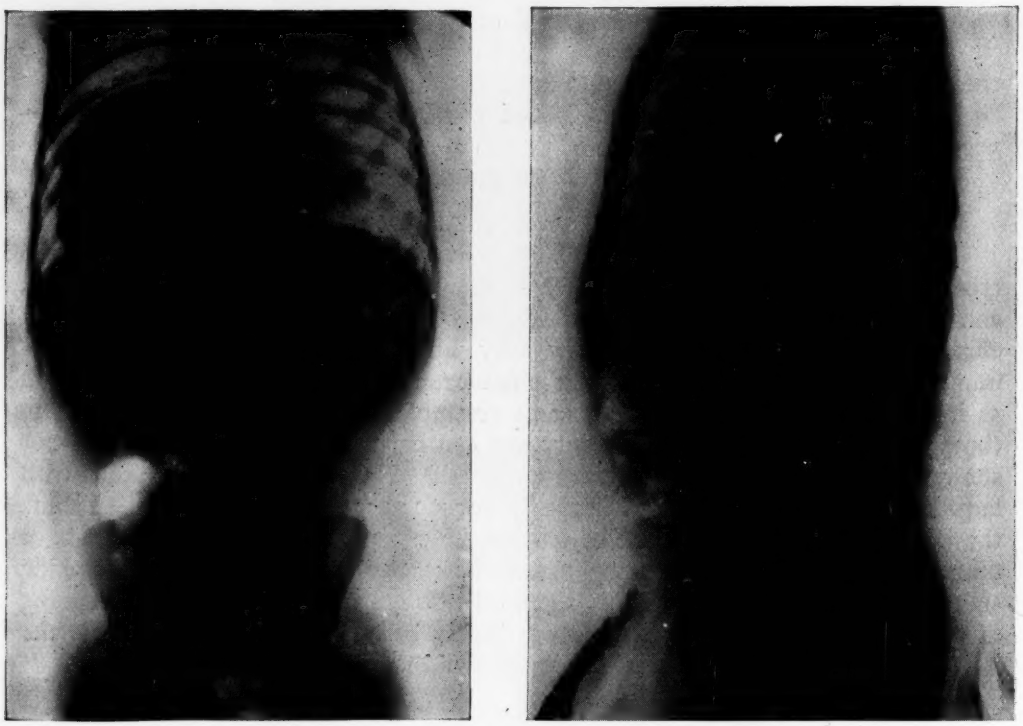
FROM THE SURGICAL UNIT, ST. THOMAS'S HOSPITAL, LONDON

ALTHOUGH cases of diaphragmatic hernia are now recognized as being comparatively common, this case is recorded as it presents certain unusual features.

The patient was a female child, who was first brought to hospital in February, 1935, at the age of nine months. Breast-feeding had been abandoned after two months, and since then attacks of vomiting had occurred with increasing frequency and severity. Her weight at the age of nine months was 12½ lb. Her diet was carefully adjusted, but the attacks of vomiting continued, and her weight on admission in May, 1935, at the age of 12 months, was only 13½ lb. Large vomits occurred

about twice a day, and the child was only able to take liquids and then very slowly; altered blood was present in both vomit and fæces. No abnormal signs were detected in the abdomen, but a radiological examination after a barium meal showed that part of the stomach was situated in the posterior mediastinum to the right of the vertebral column (*Fig.* 422). After two months' careful dieting in the ward, it was apparent that no improvement had occurred or could be expected.

FIRST OPERATION.—Laparotomy was performed by Mr. Max Page on July 10, 1935, the child then being fourteen months old; the pylorus and the greater part of the stomach were reduced from a hernial sac in the neighbourhood of the œsophageal opening and an attempt was made to repair the opening by catgut



A B

FIG. 422.—Radiograph after barium meal, showing the greater part of the stomach in the posterior mediastinum. A, Antero-posterior view; B, Lateral view.

sutures placed across the orifice. The child made a good recovery, vomiting ceased, and weight was put on rapidly. This improvement lasted for five months, although radiological examination one month after the operation showed that the hernia had recurred.

In December, 1935, the child was readmitted owing to recurrence of severe vomiting for four days; the weight was then 19 lb. Radiological examination showed that the hernia was present. Vomiting continued and the child rapidly lost weight.

SECOND OPERATION.—On Jan. 15, 1936, a second laparotomy was performed by Mr. Max Page. The pylorus and the greater part of the stomach were again reduced from the hernial sac with some difficulty; the presence of adhesions

prevented any attempt at repair in so feeble a subject. Gastropexy was performed by suturing the greater curvature of the stomach to the anterior abdominal wall. The child's post-operative condition was poor; vomiting continued, and she died on the third post-operative day.

POST-MORTEM EXAMINATION.—The hernial sac was empty, the stomach was attached to the anterior abdominal wall, and no leakage or obstruction was apparent. The hernial orifice was caused by gross dilatation of the œsophageal orifice in the diaphragm; the œsophagus was short, so that the upper third of the stomach was always above the diaphragm; sections taken from the thoracic portion of the stomach prove that this is stomach and not a dilated œsophagus. The anterior surface of the stomach was attached to the anterior margin of the œsophageal opening, and the sac of the hernia, consisting of the upper part of the lesser sac

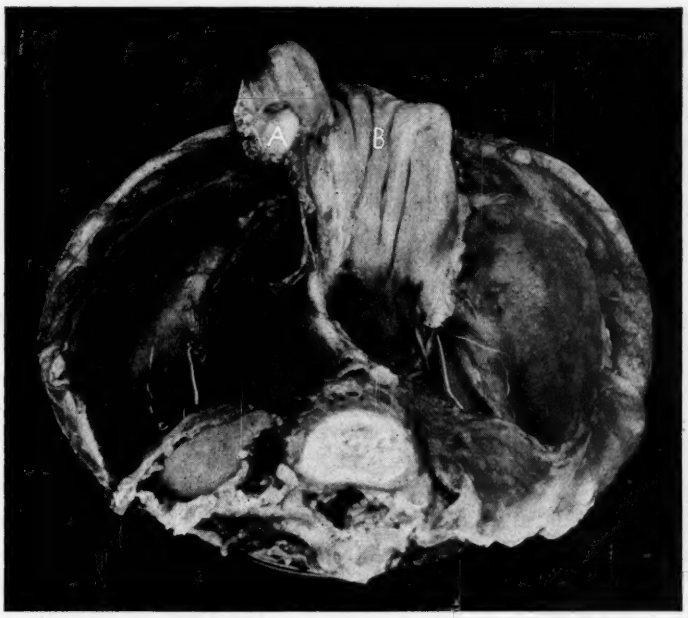


FIG. 423.—Diaphragmatic hernia. Hernial orifice seen from below.
A, Pylorus; B, Posterior aspect of stomach.

of the peritoneum, passed upwards behind the stomach into the posterior mediastinum on the right of the vertebral column (*Fig.* 423). Before operation the greater curvature of the stomach and the pylorus had rotated upwards into this hernial sac. The œsophageal opening caused no constriction of the stomach, and the child's symptoms were due to this rotation of the greater curvature and the pylorus through the orifice into the sac.

The œsophageal orifice of the diaphragm, although dilated, had normal boundaries, and using Dunhill's classification this represents an extreme example of the short-œsophagus type of diaphragmatic hernia.

The points of interest are:—

1. The sac was formed entirely from peritoneum of the lesser sac. The anterior wall of the stomach was attached firmly to the anterior margin of the œsophageal opening. There was no sac passing into the thorax in front of the stomach.

2. The sac passed into the posterior mediastinum entirely to the right of the vertebral column.

3. The possibility of gastropexy proving a satisfactory procedure.

In this case the obstructive symptoms were due to rotation of the greater curvature and pylorus through the sac into the mediastinum and not to the fact that the upper third of the stomach was situated permanently in the thorax.

OBSERVATIONS BY MR. C. MAX PAGE.—

The specimen described is unusual in the large size of the hernial sac extending into the right pleural area. Operation became imperative at an early age on account of the severity of the symptoms caused by the herniation of a large section of the stomach. A radical cure of the hernia by the thoracic route did not seem justifiable in this infant. It would, however, appear possible that if a fixation of the stomach had been carried out at the first operation the child might have survived. It seems likely that an effective fixation of the distal half of the stomach would prevent prolapse of sufficient of that viscus into the hernial sac to produce symptoms.

I wish to thank Mr. C. Max Page for permission to publish this case, which was under his care, and to express my indebtedness to Sir Thomas Dunhill for his assistance in interpreting the findings.

AN UNUSUAL VASCULAR COMPLICATION OF CERVICAL RIB

BY E. R. FLINT

SURGEON TO THE GENERAL INFIRMARY, LEEDS

NELLIE A., aged 45, was admitted to the General Infirmary at Leeds on May 11, 1936, under Dr. McAdam, who obtained from her the following history.

HISTORY.—Except for poor circulation all her life, in the hands particularly, there was nothing of much note until two months before admission, when she noticed an aching pain in the right thenar eminence; the pain passed into her wrist and palm, and more recently upwards into the shoulder. She had no similar pain in her left arm or anywhere else. During the two months she had slight numbness in the right index finger. For several years she had noticed a small pulsating lump in the right supraclavicular fossa. On Sunday, May 17, it was observed that the pulsation had disappeared—it did not re-appear again—and on the same day the lump became painful and much larger, and the pain in her hand worse.

ON EXAMINATION.—Examination five days before operation showed an ill-defined swelling filling the right supraclavicular fossa; it was tender and fixed deeply, but not to the skin. There was no pulsation in or over the lump. Palpation caused local pain, but no radiation. There was no pulsation in any of the vessels of the right arm; on the left side pulsation was normal. There was slight wasting of the muscles of the arm and hand on the right side and some diminution in muscular power. Sensation was normal and equal on the two sides; the colour of the right hand was rather bluer than on the left side. No other abnormal physical signs were present.

X rays showed a cervical rib on both sides, apparently complete.

Dr. McAdam asked me to see her with a view to surgical treatment.

OPERATION (May 25).—After dividing the skin and platysma (*Fig.* 424) a yellowish-white non-pulsating tumour presented external to the scalenus anticus. It was about the size of, and shaped very like, a Victoria plum, and extended a little

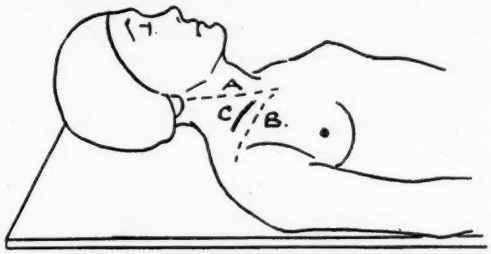


FIG. 424.—Showing position and shape of incision. The sternomastoid is indicated by the broken line A, the clavicle by B; C shows the incision.

beneath the scalene (*Fig.* 425). A small incision was made into it and some clot evacuated. The tumour lay on a cervical rib, which had a keen anterior edge, and at one place a rather sharp projection. The rib was complete; that is to say, it was united to the first rib by bone, and ran a fairly straight course between its vertebral and costal attachments. The scalenus anticus was divided, and the

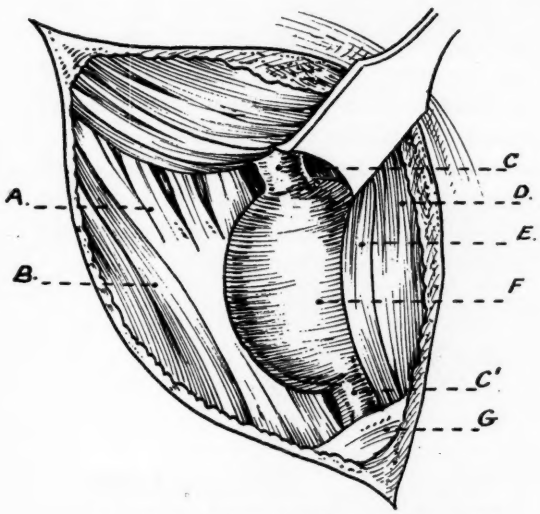


FIG. 425.—Condition found on exposure, with the tumour in situ. A, Brachial plexus; B, Scalenus medius; C and C', Subclavian artery; D, Sternomastoid; E, Scalenus anticus; F, Tumour; G, Clavicle.

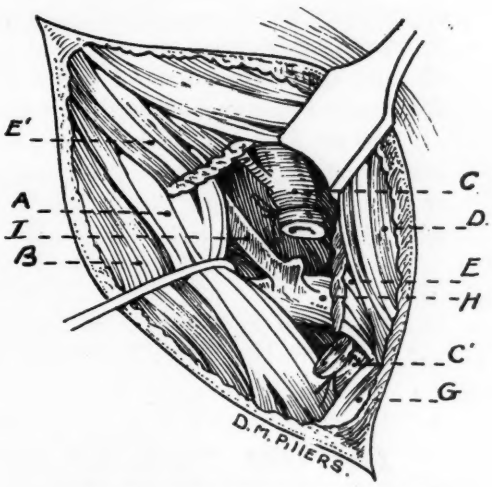


FIG. 426.—After ligature of subclavian artery and removal of tumour. A, Brachial plexus, held aside; B, Scalenus medius; C and C', Subclavian artery ligatured; D, Sternomastoid; E and E', Scalenus anticus divided; G, Clavicle; H, 1st rib; I, Cervical rib (note sharp edge).

subclavian artery was seen entering and leaving the swelling; ligatures were placed above and below the tumour and it was removed, the rib being left in situ (*Fig.* 426). The patient was discharged on June 5 in good condition.

On July 24 the patient was seen again. All pain has gone, and she is doing house-work, which she had been unable to do for several weeks before operation. The hand is a good colour.

PATHOLOGICAL REPORT (Prof. M. J. Stewart).—Sections have been taken both across the rupture in the wall of the artery and across the non-ruptured portion. Actually the breach in the arterial wall does not suggest an aneurysm, but rather a sudden rupture of an arterial wall which had been weakened from without. The lumen is lined by organizing thrombus of some standing, and the middle coat shows much diffuse fibrosis, but the chief changes are in the adventitia and external to it, where there is a broad zone of active granulation tissue. This is in keeping with the suggestion that pressure of a cervical rib may be the prime etiological factor. None of the changes suggest a syphilitic arteritis, although the peri-arteritis might conceivably be syphilitic in nature. This, however, seems unlikely, since the middle coat shows no such change.

Commentary.—Many of the features presented by this case conform to the usual clinical experience: for example, the patient was a middle-aged woman with an accessory rib on both sides (about 75 per cent of cases) but symptoms only on the right side. The unusual thing about this patient is the type of vascular complication, which was apparently a false aneurysm brought about by the constant pressure of an unusually sharp-edged rib. Thrombosis in the subclavian artery has been seen fairly often, and aneurysm has been reported (Billington[1] records a fusiform aneurysm in association with a cervical rib), but I have been unable to find any description in the literature of a case such as is here described.

Two causative factors appeared to be present: (1) The almost cutting edge of the rib; (2) Its nearly straight course, giving the scalenus anticus the fullest opportunity for constricting the artery. Telford and Stopford[2] find it difficult to believe that pressure is exerted on the subclavian artery in the presence of a cervical rib; the case reported above would seem to make it quite certain that such pressure does occur sometimes, though it is not the cause of all the symptoms.

No attempt was made to remove the rib in my case, as I believe this to be unnecessary and certainly more mutilating than simple division of the scalene at or near its attachment to the first rib; such a division opens up the space at the root of the neck quite enough to accommodate all the structures freely.

REFERENCES

[1] BILLINGTON, W., *Brit. Jour. Surg.*, 1931, xix, 334.
[2] TELFORD, E. D., and STOPFORD, J. S. B., *Ibid.*, xviii, 557.

THYROID TUMOUR WITH MULTIPLE METASTASES

BY JOHN WILLIAMSON
MEDICAL OFFICER, TANGANYIKA TERRITORY

THIS case was seen by me in 1932, soon after W. K. Connell had published a paper in the BRITISH JOURNAL OF SURGERY[1] on two cases of thyroid metastases seen by him in Dar-es-Salaam.

I do not think there is any doubt but that this is a case of thyroid metastases, but it differs from Dr. Connell's cases in that the metastases are multiple and the enlargement of the thyroid is very marked.

Unfortunately my notes on the case are not available, and the following very brief history has had to be supplied from memory.

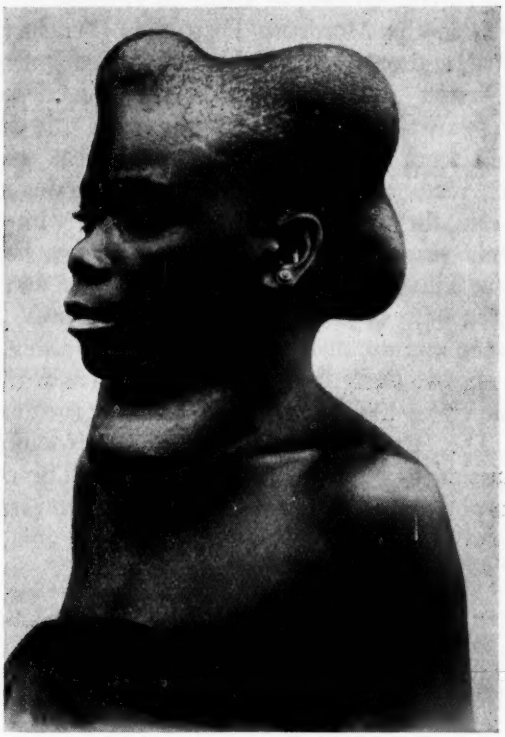


FIG. 427.—Photograph of the patient showing the thyroid enlargement and the growths in the skull.

FIG. 428.—Radiograph of the skull.

The patient was a female African, of about 26 years of age, and she came from a village near Turiani, in the Morogoro District of Tanganyika Territory. She was a healthy individual, and only came to Morogoro Hospital because she was seen by a keen African District Sanitary Inspector, and his description aroused my curiosity. Histories are always difficult in Africans, and this case was no exception to the rule. It would seem fairly certain, however, that the growths had been present for at least ten years, and that the rate of growth was very slow. Some of her friends actually said they could not remember her without them. She was married to a man many years her senior, and had had two healthy children, both of whom died in early childhood. The Wassermann reaction was ++.

There was nothing of note on examination. The pulse was regular in rate and rhythm, was 70 per minute, and responded normally to exercise.

The photograph shows clearly the healthy appearance of the patient (*Fig.* 427), and the radiograph the crater-like condition of the new growths (*Fig.* 428).

I have to thank The Honourable The Director of Medical Services, Dar-es-Salaam, for his permission to publish the illustrations. The radiograph was taken by Mr. J. E. Brunnen, of the Tanganyika Civil Service, and intensified by Messrs. Kodak Ltd.

REFERENCE

[1] CONNELL, W. K., " Thyroid Metastases in Bone ", *Brit. Jour. Surg.*, 1929–30, xvii, 523.

REVIEWS AND NOTICES OF BOOKS

Techniques Chirurgicales. By A. GOSSET, Chirurgien de la Salpêtrière, Professeur de Clinique chirurgicale à la Faculté de Médecine de Paris; with the collaboration of MM. L.-G. AMIOT, IVAN BERTRAND, JEAN CHARRIER, P. FUNCK-BRENTANO, J. GARCIA-CALDERON, JEAN GOSSET, P. HAUDUROY, R. LEDOUX-LEBARD, R. LEIBOVICI, G. LŒWY, P. PETIT-DUTAILLIS, P. ROUCHÉ, R. SAUVAGE, G. SEILLÉ, R. SOUPAULT, M. THALHEIMER, and E. WALLON. Imperial 8vo. Pp. 434 + viii, with 219 illustrations. 1936. Paris: Masson et Cie. Paper covers, Fr. 105; bound, Fr. 125.

HERE M. Gosset and his colleagues present an account of the Surgical Service in the Salpêtrière Hospital in Paris, of which M. Gosset has been the Director for more than twenty years.

In addition to a number of technical articles, e.g., on Diaphragmatic Hernia and Intestinal Obstruction, which describe the activities of the Clinic, Gosset enunciates his matured convictions concerning the housing, organization, and staffing of a surgical unit. The organization comprises both the Clinic of the Faculty and also an Anti-cancer Centre such as exists in Paris and in some of the large French towns. The number of beds in the Clinic is 300, which are divided among pavilions, each containing 30 beds. To each pavilion two assistants are attached who work under the authority of the "chef de service". The Clinic includes numerous assistants, both surgical and medical, and a complete service of radiology, radiotherapy, curie-therapy, and laboratories of pathology, chemistry, bacteriology, and hæmatology. This contribution should be of great service to anyone contemplating the organization of a surgical unit.

Minor Surgery. By FREDERICK CHRISTOPHER, S.B., M.D., F.A.C.S., Associate Professor of Surgery at the Northwestern University Medical School, Chicago; Chief Surgeon at the Evanston (Ill.) Hospital. With a Foreword by ALLEN B. KANAVEL, M.D., F.A.C.S., Professor of Surgery at the Northwestern University Medical School. Third edition, reset. Large 8vo. Pp. 1030, with 709 illustrations. 1936. London and Philadelphia: W. B. Saunders Co. 42s. net.

MINOR SURGERY is defined here as the surgery which has a low mortality; which requires but few assistants; and which is done generally in the hospital out-patient department or in the office.

Though dealing with minor surgery it is not a little book, for it is an octavo volume of 1030 pages. Nor does it treat the subject as one of minor importance. Rather it emphasizes the point that minor surgery may become major surgery through carelessness or incompetence, and that there cannot be too much instruction in minor surgery.

The whole range of the subject is treated most exhaustively and sensibly, and it should prove a useful book of reference to all students beginning surgery and also to resident medical officers.

One of the best sections in the opinion of the reviewer is that dealing with the treatment of wounds and the methods of securing early healing and restoration of function.

Manual of Emergencies: Medical, Surgical and Obstetric: Their Pathology, Diagnosis and Treatment. By J. SNOWMAN, M.D., M.R.C.P.Lond. Third edition. Crown 8vo. Pp. 401 + ix. 1936. London: John Bale, Sons & Danielsson Ltd. 10s. 6d. net.

IT is very remarkable how much useful information has been packed into this small manual. It was originally based on the treatise of Lenzmann, but has been well adapted to the modern standard of teaching in British text-books. After treating of shock, the various emergencies which may affect the respiratory, cardiac, nervous, alimentary, and urinary systems are dealt

with. In the abdomen acute general peritonitis is discussed before the causative conditions, e.g., appendicitis, and we think that this is a mistake. Fifty pages are devoted to poisons, and the book concludes with a consideration of obstetrical emergencies. The manual is clearly written and its information accurate and reliable.

Operative Surgery. By ALEXANDER MILES, M.D., LL.D., F.R.C.S. (Edin.), Consulting Surgeon, Royal Infirmary, Edinburgh, and D. P. D. WILKIE, M.D., F.R.C.S. (Edin. and Eng.), Professor of Surgery, University of Edinburgh. Second edition. Demy 8vo. Pp. 631 + xx, with 329 illustrations. London: Oxford University Press. 21s. net.

THIS is the second edition of a text-book on operative surgery, the first edition of which was reviewed in this JOURNAL. The book is intended for students attending a course of operative surgery. It is well illustrated, and sets out in lucid manner all the operations which a student is expected to know.

The Surgical Technic of Abdominal Operations. By JULIUS L. SPIVACK, M.D., Assistant Professor of Surgery, University of Illinois College of Medicine; Professor of Operative Surgery and Surgical Anatomy, Cook County Graduate School of Medicine; etc. $10\frac{1}{2} \times 7\frac{1}{4}$. Pp. 718 + xvi, with 362 illustrations. 1936. Chicago: S. B. Debour.

IN a volume of some size the author describes with meticulous detail the surgeon's technique within the abdomen. No attempt is made to give indications for the procedures described; he deals with the *How*, not the *Why*. Starting with the simplest procedures of knot-tying and sutures, he builds up the detailed steps of typical abdominal operations. The field is covered very thoroughly, and some of the procedures are of historical rather than of practical interest.

In spite of the limitations necessarily inherent in a book of this type, it has a very definite place in the surgical library, both for the post-graduate student of surgery and for the man of maturer years, and one has only to dip into its pages to realize how relatively narrow a groove of technique one is apt to develop and to hand on to one's assistants. Whilst apprenticeship is essential, it is the richer for the study of surgical technique as a basic factor in the craft of surgery. The illustrations are numerous and give reality to every operative step described in the text, whilst a full bibliography rounds off each chapter.

Appendicitis: When and How to Operate. By W. J. STEWART MCKAY, M.B., B.Ch., B.Sc., Senior Surgeon, Lewisham Hospital, Sydney. Demy 8vo. Pp. 260 + xii, with 16 illustrations. 1936. Sydney: Angus & Robertson Ltd. 12s. 6d. net.

IT is hard to conceive that in these days any medical man, even in the wilds of Australia, trained as he must have been at a school of medicine of some University, could possibly display such gross ignorance of the practical details of aseptic operative surgery as the author throughout this work assumes. Far too much space is occupied in describing the elementary minutiæ of the requisite preparation for operation, and much of it might well have been left to the practitioner's resourcefulness and ingenuity. The book is stated, in the Preface, to have been started as a guide to the general practitioner in the country who "thinks that he would like to try and remove a diseased appendix". If his ignorance be anything approaching what the author would have us believe, it would, we consider, be very much better to discourage rather than encourage him to undertake "His First Appendix Operation", which was the title originally intended for this work.

The author has, himself, evidently had a ripe experience in the diagnosis and treatment of appendicitis—such an experience as commands respect for his opinions, although some of these are distinctly unorthodox. He believes that "chill is the chief cause of appendicitis in those persons who are predisposed to catarrh by the inheritance of exuberant lymphoid tissue in the appendix, which is almost invariably accompanied by an exuberance of lymphoid tissue in Waldeyer's circle in the nasopharynx", and that "children and adults who have enlarged tonsils always have a large appendix: people who have a large appendix are often subject to catarrh of the colon and appendix".

In the clinical investigation of a case of acute appendicitis the importance of vaginal examination in a married woman is very rightly stressed, but no mention is made of the equally important rectal examination in males and young females. In describing rigidity of the abdominal wall no proper distinction is drawn between 'voluntary' and true 'involuntary' rigidity, nor is their differential diagnosis discussed. On the whole the technical details of the operative procedures recommended for the different types and stages of acute appendicitis are admirably described and are such as we can commend—with a few exceptions. Thus, in operating for appendicitis with diffuse peritonitis it is advised (p. 201) that after removal of the appendix a fresh opening should be made in the median line and the peritoneal cavity well flushed out with warm saline solution—a practice we had thought quite obsolete; while in opening an appendicular abscess transperitoneally the protection of the peritoneal cavity by gauze packing—a very important matter—is very inadequately described. The advice given as to the non-removal of the appendix in large abscess cases is extremely sound, all the more so when proffered to one undertaking "his first appendix operation". Several good recipes and some useful hints on diet are given in an appendix.

The Thyroid: Surgery, Syndromes, Treatment. By E. P. SLOAN, M.D. Edited by Members of the Sloan Clinic. With a Foreword by WM. SEAMAN BAINBRIDGE, M.D. $10\frac{1}{4} \times 7\frac{1}{4}$. Pp. 475 + ix, with 99 illustrations. 1936. London: Baillière, Tindall & Cox. 45s.

THIS volume makes no pretence of being an encyclopædic work of reference, but embodies the working theories and conclusions, unhappily posthumous, of a surgeon who devoted twenty-five years of his life exclusively to goitre work. That his practical experience was abundant is evident from the statement that at the time of publication some 15,000 cases of goitre had been operated on in the Sloan Clinic. There is, indeed, evidence in the book that the vast amount of routine operative work has militated against scientific investigation to some extent, for the sections dealing with the details of operative technique and surgical anatomy are more convincing than those concerned with etiology and pathology. The author elaborates a theory that there is in secondary toxic goitre an 'anaphylactic group' of cases in which such symptoms as urticaria and asthma are due to absorption of certain unspecified degeneration products. No attempt, however, appears to have been made to establish this interesting theory upon a sound experimental basis. The theory that toxic goitre is a dysthyroidism rather than a hyperthyroidism is supported, and attention is concentrated on the thyroid gland, with little consideration of the part played by the hypothalamus and the pituitary in initiating the hyperplastic change in the thyroid. There is no mention of Harington's important researches on thyroxine. The proof-reading leaves much to be desired, and it is irritating to find 'septæ' recurring as the plural of septum, and to be told that "the thymus measures 5 c.c. in length and 4 c.c. in width".

Die Nachbehandlung nach Operationen: ein Lehrbuch in Vorlesungen. By Prof. Dr. PAUL REICHEL, Geheimer Sanitätsrat. Third edition. Large 8vo. Pp. 499+xii, with 85 illustrations. 1936. Munich: J. F. Bergmann. Paper covers, RM. 24; bound, RM. 25.80.

THIS book, which is in the form of forty-two lectures, is of especial value in describing and discussing the after-treatment of operation cases because it represents the matured experience of its distinguished author. The first edition was written in Breslau in 1896, when he had only ten years' experience as an assistant but was filled with the enthusiasm of youth. The second was from Chemnitz in 1908, and the present, in 1936, when he has had more than thirty years' experience as the director of a large hospital in Munich.

The work is divided into two parts: the first, of fourteen lectures, deals with general surgical problems, concluding with a discussion of the role of physiotherapy, in which mechanical apparatus plays a larger part than we are accustomed to assign to it in this country.

The second part, consisting of twenty-eight lectures, deals with all the organs of the body, and special surgical departments. Of these no less than eight are concerned with laparotomy and operations on the stomach and intestines. The last eight lectures deal with the after-treatment of injuries and operations on the limbs. A specially useful feature of this section is the discussion about prognosis and insurance liabilities.

Chirurgische Operationslehre. By Dr. MAX SAEGESSAR, Privatdozent an der Universität Bern. Crown 8vo. Pp. 189 + vi, with 132 illustrations. 1935. Berlin: Julius Springer. Paper covers, RM. 7.50; bound, 8.60.

THIS short and compact book is primarily a guide to an operation course on the cadaver. As it represents the teaching of the famous Bern School, and is introduced by De Quervain, its authority is beyond question. Its main value is the many anatomical diagrams, which are models of accuracy and clearness. As in all books of this kind, the space devoted to the ligature of arteries and amputations is out of all proportion to their importance in clinical surgery.

Recent Advances in Genito-urinary Surgery. By HAMILTON BAILEY, F.R.C.S. (Eng.), Surgeon, Royal Northern Hospital; Surgeon and Urologist, Essex County Council; and NORMAN M. MATHESON, M.B., F.R.C.S., M.R.C.P., Surgeon, Central Middlesex County Hospital. Large post 8vo. Pp. 213 + viii, with 89 illustrations. 1936. London: J. & A. Churchill Ltd. 15s. net.

THIS is one of the latest additions to the "Recent Advances" series, and it is no mean praise to state that it keeps up to the standard of the previous volumes. It is well written and attractively published; in it the authors more than justify their attempt to place before their readers recent innovations as they affect the commoner conditions encountered in urological practice.

Amongst the many excellent discussions we should select that on the etiology of urinary stone as one of the best; it embodies all the most recent work on the subject and lays emphasis on the undoubted relation between calculus formation and endocrine dysfunction; since renal colic and hæmaturia may antedate the clinical manifestations of hyperparathyroidism, it seems probable that some, at any rate, of those disheartening cases of recurrence of stone in the kidney may owe their cause to this disease.

On two points we venture to suggest that the authors do not express the current opinion of urologists; they do not mention the median subumbilical incision for the removal of stone from the pelvic ureter—a surprising omission—and they assert that most surgeons now remove the ureter "in its entirety" when excising a tuberculous kidney; we own that they add that this technique is not universally agreed on, but, if it be done with the object of avoiding a subsequent sinus in the loin, we believe that it is unnecessary.

There are excellent sections on pyelography, on renal function tests, on the role of sympathectomy in urology, and on modern methods of treating the enlarged prostate.

The numerous illustrations are hardly up to the high standard to which Mr. Hamilton Bailey has accustomed us, but this may be explained by the fact that they have been borrowed. The bibliography at the end of each section is admirable, and is exactly what is needed by the busy surgeon.

This handy little book contains a vast amount of useful and interesting information, and we have no hesitation in recommending it to the readers of this JOURNAL.

Surgical Diseases and Injuries of the Genito-Urinary Organs. By Sir JOHN THOMSON-WALKER, D.L., M.B., C.M.Ed., F.R.C.S.Eng., Consulting Urologist and Emeritus Lecturer in Urology, King's College Hospital, etc. Second edition, revised: edited by KENNETH WALKER, M.A., M.B., B.C.(Cantab.), F.R.C.S..Eng., Jacksonian Prizeman and Hunterian Professor, R.C.S., 1911, 1922, 1924, 1933; Lecturer in Venereal Diseases, St. Bartholomew's Hospital; etc. Medium 8vo. Pp. 974 + xviii, with 283 illustrations and 25 colour and 33 black-and-white plates. 1936. Cassell & Co. Ltd. 32s. 6d. net.

IN a review of the first edition of this book, in 1914, we ventured to predict that it would quickly take its place as a standard text-book in urology; this has been verified, and the work has proved of the greatest service to the general surgeon as well as to the specialist.

This new volume is a credit to the authors, the publishers, and the artists concerned; it is well bound and clearly printed, and the illustrations are not only well reproduced and of artistic merit, but they are of real help in elucidating the text.

Owing to the advances in genito-urinary surgery during the last twenty years it has been found necessary to write new chapters on renal function tests, on transurethral operations,

on obstruction at the bladder neck, and on impotence and sterility; these have been admirably done, and give a clear and up-to-date account of our present knowledge of these subjects. For the same reason those sections which treat of prostatic enlargement, of renal tuberculosis, and of bladder tumours have had to be rewritten; the subject of the use of X rays in diagnosis and treatment has been dealt with more fully, and the recent developments in pyelography have necessitated many additions to both the text and the illustrations.

We cannot help thinking that the numerous readers of this rather monumental text-book will feel a sense of disappointment, mingled with a mild surprise, when they eagerly turn to certain parts to find out what two such well-known urologists have to say on some of the very latest advances in treatment.

For instance, we not unnaturally looked up the treatment of the *Bacillus coli* infections by the use of mandelic acid; we own to feeling slightly shocked when we found no mention of this drug either in the text or the index. In the same way, we sought in vain for any reference to the treatment of retention of the testis by endocrine therapy.

It may quite well be that the authors consider that the introduction of mandelic acid is too recent for its value to be properly assessed, and that they disapprove of the use of injections which favour the descent of the testis, possibly by bringing about a premature development of the secondary male characters. We think, however, that the student of surgery would expect to find in a modern volume of urology a discussion on such important points and some guidance as to their value; moreover, at the higher examinations in surgery, a candidate would surely be required to know something on such subjects.

We hesitate to criticize the chapter on tumours of the suprarenal gland, for it might be urged that this organ belongs neither to the urinary nor to the genital apparatus; since, however, several pages have been devoted to these neoplasms we record our surprise that it has not been brought up to date. The astounding changes that may occur in the female afflicted by such a growth—the moustache and beard, the apical baldness, the general hirsuties, the amenorrhœa, the rise of blood-pressure, and the alteration in the sexual outlook—are not mentioned; no reference is made to the late Sir Percy Sargent's successful case of operation, nor to any recent literature on the subject, though several cases have been published in the last few years. Indeed, with the exception of a reference to an article by Broster in 1931, the bibliography at the end of this chapter is quite out of date.

An excellent chapter on Stone in the Ureter is similarly marred by what we think must have been an oversight of the authors. In discussing the operative treatment of stone in the pelvic ureter they state that "the median subumbilical incision and extraperitoneal approach is far superior to the lateral curved incision": a remark with which every surgeon who has tried the former operation will most emphatically agree. It is disconcerting, therefore, to find under the heading of operations on the ureter, a long description of what is admittedly the inferior technique and a quite inadequate account of that previously recommended; it is, in fact, dismissed in some seven lines, which would be of very slender assistance to any surgeon who was contemplating carrying out such an operation.

Apart from these minor defects, which we recognize as liable to be included in any work of this magnitude, we have nothing but praise for the book and we cordially commend it to our readers.

In the inevitable future third edition we hope the authors and publishers will have mercy on the poor reader and issue the work in two, or even three, volumes; this *opus ponderosum* weighs just over five pounds, and it is impossible to read it in comfort.

Surgery for Dental Students. By PHILIP H. MITCHINER, M.D., F.R.C.S., C. E. SHATTOCK, M.D., F.R.C.S., E. G. SLESINGER, B.Sc., F.R.C.S., and CECIL P. G. WAKELEY, D.Sc., F.R.C.S. 9 × 6. Pp. 364 + ix, with 105 illustrations. 1936. London: Baillière, Tindall & Cox. 12s. 6d.

THE authors aim particularly at covering the Syllabuses in Surgery and Surgical Pathology of the Royal Colleges of England, Scotland, and Ireland, and of the British Empire Universities: they do more, as they have succeeded in producing a concise and practical text-book for dental students.

The general surgical field is touched with a light hand, yet with sufficient detail to give the student a working insight into underlying pathological and clinical processes; coming

to the territories where a working knowledge of surgery is of importance the teaching becomes fuller and more detailed, and is of great practical value in dealing with the problems common to the surgeon and dentist.

The skill with which condensation has been carried out without loss of efficiency should make the book popular with students and a convenient manual for reference after qualifying.

Diagnostik mit freiem Auge Ektoskopie und Ektographie mit einem Anhang neuere Tast-befunde. By Dr. EDUARD WEISZ and Dr. L. SCHMIDT. Fourth edition. Royal 8vo. Pp. 176 + viii, with 84 illustrations. 1933. Berlin and Vienna: Urban & Schwarzenberg. Paper covers. RM. 9.50; bound, RM. 11.

THIS monograph is a description of how much can be learned by mere inspection with the naked eye and by palpation. In the thorax the movements of the intercostal muscles, and in the abdomen the movements of the abdominal muscles, on respiration and on speaking are described in great detail, and especially as these movements are affected by various morbid conditions. In the present day, when so much reliance is placed upon diagnosis by laboratory and instrumental methods, it is well to be reminded of how much can be learned by the trained eye and finger.

Gestes et Procédés techniques de Chirurgie Générale. By JEAN BERGER, Chirurgien des hôpitaux de Paris. 9¼ + 6¼ in. Pp. 138, with 124 illustrations. 1936. Paris: Masson et Cie. Fr. 32.

THIS is a refreshingly original book. It is not a text-book of operative surgery, but a manual dealing with surgical technique. It describes not so much what to do as how to do it. It shows how to hold a pair of scissors and to cut a ligature; how to handle a needle, and to put on a pair of forceps; and a hundred other points of technique. It is good, and will repay study by any surgeon who thinks about, and wishes to polish, his methods.

A Descriptive Atlas of Radiographs. An Aid to Modern Clinical Methods. By A. P. BERTWISTLE, M.B., Ch.B., F.R.C.S. (Edin.). Third Edition, revised and enlarged. Cr. 4to. Pp. 560 + xxxi, with frontispiece and 794 illustrations. 1936. London: Henry Kimpton. 42s.

THE fact that a third edition of this book has been called for in ten years shows that its value has been appreciated.

It consists of 794 cases in which an X-ray print is accompanied by a short description and sometimes a clinical history. In the present edition two new sections appear, one on medico-legal problems, chiefly the determination of the age of the fœtus, and one on anthropology, which contains only two figures, one of which is very obscure. In his introductory remarks the author makes a well-argued appeal for a museum of radiography. As a book of reference it is invaluable, but its usefulness will be improved as time goes on by the inclusion of more radiographs of difficult cases, whilst the obvious things might be omitted. For example, in the section on fractures, there is only one figure of a fractured os calcis, whilst many of the common fractures of the shafts of the long bones hardly need the wealth of illustration given to them.

Clinical Handbook for Residents, Nurses, and Students. Being the Routine Methods of St. Vincent's Hospital, Sydney. By Members of the Staff of the Hospital. Edited by VICTOR M. COPPLESON, Ch.M., F.R.C.S., F.R.A.C.S., and DOUGLAS MILLER, Ch.M., F.R.C.S., F.R.A.C.S. Cr. 8vo. Pp. 205 + x. 1936. London and Sydney: Angus & Robertson Ltd. 6s.

IT is obvious that a manual of this sort compiled by the members of a hospital staff for the guidance and direction of their own students, nurses, and residents, must be of great practical value.

The chapters on clinical pathology and biochemistry, giving as they do an explanation and description of all ordinary methods, appear to us to be of the greatest value. Also the

many reference tables, e.g., those of the normal content of various substances such as urea, calcium, etc., in the blood, are most useful. It is curious that whilst the management and nursing of ophthalmic, E.N.T., skin, and gynæcological cases is described, there is no section on the management and nursing of general surgical or fracture cases, nor of the technique of radium application. The opening section on sterilization is excellent, but we think it illogical to describe a special '4-day' method for operations involving bones and joints, which implies that the method used for other cases is inadequate. The method of cutting off plaster casts with a knife seems unnecessarily risky.

Baillière's Synthetic Anatomy. By J. E. CHEESMAN. Part XIII. The Eye and Orbit. Plates 7½×9 in. 1936. London: Baillière, Tindall & Cox. 3s. net. Complete set of 14 parts in Binder, 45s. net. Loose Leaf binding case, to take whole work, 3s. net.

WE have already given an appreciative review of the early parts of this work, in which we described it as "the best and most convenient substitute for the body". The last section, on the orbit, consisting of eight coloured plates, completes the series. It consists of superimposed coloured pictures on a transparent medium, which allows each layer to be seen through the one above it.

The facts that the work has been produced in French, German, and Spanish, and that over 100,000 copies have been issued, are sufficient evidence of its proved value in the teaching of anatomy.

Text-book of Pathology. By Sir ROBERT MUIR, M.A., M.D., Sc.D., LL.D., F.R.S., Professor of Pathology, University of Glasgow; Pathologist to the Western Infirmary, Glasgow. Fourth edition. Medium 8vo. Pp. 994 + viii, with 571 illustrations. 1936. London: Edward Arnold & Co. 35s. net.

THIS is a new edition of the well-known text-book on pathology, and no further notice seems to be required than the statement that the incorporation of important additions to knowledge that have occurred since the last edition ensures that the book remains the standard work in pathology in this country.

Die Wirbelgelenke. By Prof. Dr. MAX LANGE, Director of the Orthopædic Clinic, Munich. Second enlarged edition. Large 8vo. Pp. 138 + viii, with 94 illustrations. 1936. Stuttgart: Ferdinand Enke. RM. 10.

THIS is a very careful study of the anatomy, physiology, and pathology of the intervertebral joints. The difficulties of accurate X-ray examination are stressed, and clear directions given for overcoming these. Then in great detail all conditions of altered position (e.g., scoliosis or kyphosis), and inflammatory, infectious, and traumatic conditions are described and figured from pathological specimens and radiographs. The greatest attention is paid to the changes in the joints between the articular processes rather than to those in the intervertebral discs. The book does not deal with problems of treatment.

Roentgendiagnostik der Knochen- und Gelenkkrankheiten. By Professor Dr. ROBERT KIENBÖCK (Vienna). Part 4. Degenerative Wirbelsäulenerkrankungen. Large 8vo. Pp. 229–450, with 214 illustrations. 1936. Berlin and Vienna: Urban & Schwarzenberg. RM. 24.

THIS is a very detailed account of the changes which take place in the vertebral bodies in chronic diseases of the vertebral column. It is chiefly concerned with static kyphosis, scoliosis, and spondylitis deformans. Its chief value lies in the numerous X-ray pictures of different regions of the spine and at different stages of the disease, together with careful clinical histories of the cases concerned. It forms a useful companion volume to that by Professor Lange which deals with the changes in the joints.

Die moderne Finsenbehandlung. By Dr. SVEND LOMHOLT, Director of the Dermatological Clinic at the Finsen Institute, Copenhagen. Large 8vo. Pp. 64, with 52 illustrations. 1934. Berlin and Vienna: Urban & Schwarzenberg.

THIS forms but a chapter in a larger work on treatment by radiotherapy. It gives in simple language the principles underlying the therapeutic use of light, both sunlight and that of various carbon and ultra-violet lamps.

The modern Finsen lamp is a carbon-arc lamp with special lenses and filters, and it is claimed to be more efficient than the older pattern. Thirty advanced cases of lupus vulgaris are presented at different stages of treatment, showing the progress towards recovery made under the Finsen light treatment.

Behandlung der Verletzungen und Eiterungen an Fingern und Hand. By Professor Dr. M. ZUR VERTH, Privatdozent in the University of Hamburg. Second edition. $5\frac{3}{4} \times 8\frac{5}{8}$. Pp. 164 + viii, with 59 illustrations. 1936. Berlin: Julius Springer. Paper covers, RM. 8.70; bound, RM. 9.60.

TRAUMATIC surgery has assumed a much more specialized aspect in Germany than in this country. This is partly due to the system of state insurance of workmen which has made available a great mass of statistics showing the relative economic importance of different forms of injury. Over 34 per cent of all finger injuries lead to some degree of permanent disability. The amounts paid in compensation for cases of finger injuries is almost double that paid for all cases of fracture of the long bones. This little book is a clearly written and well-illustrated account of all the different types of injury and infection to which the hand and fingers are subject, and the most effective way of treating the same. It concludes with a useful discussion of the different types of amputation of the fingers and hand suitable for hand workers and brain workers.

Die Kurzwellenbehandlung in der Chirurgie. By Dr. ALFONS LOB, Roentgenabteilung der Chirurgie, Univ.-Klinik, Munich. Medium 8vo. Pp. 68. 1936. Stuttgart: Ferdinand Enke. RM. 3.60.

THIS is a short and simple explanation of the physical nature and action of short-wave therapy. The author holds that the early and exaggerated promises of the therapeutic value of this method have not been fulfilled. Especially is this the case in the treatment of acute pyogenic affections, in which short-wave therapy can do no more than act as an adjuvant to the ordinary surgical treatment. With new growths, too, the short-wave therapy has given doubtful benefit, and that chiefly when combined with X-ray treatment. The principal value of the method according to the Munich experience is in the treatment of sub-acute and chronic affections.

The Patient Looks at the Hospital. By FLORENCE G. FIDLER. With a Foreword by JOAN MALLESON, M.B., B.S. Crown 8vo. Pp. 128 + xii. 1936. London: Robert Hale & Co. 2s. 6d. net.

THIS is a useless book which illustrates the fallacy of arguing from a particular to the general. Miss Fidler appears to have had some unfortunate experience in a voluntary hospital. She deduces therefrom that matrons are martinets, nurses are fools, doctors are hoodwinked, and committees' interest lies more in the collection of money than in the welfare of the hospital inmates. She views everything with a jaundiced eye and seems to be wholly ignorant of life in a well-organized hospital. The book is dedicated "to the Subscribers to all Voluntary hospitals". Dr. Joan Malleson writes a discrete foreword.

BOOK NOTICES

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

St. Bartholomew's Hospital Reports. Edited by W. G. BALL, G. EVANS, G. GRAHAM, G. HADFIELD, C. F. HARRIS, W. SHAW, H. H. WOOLLARD, R. C. ELMSLIE, J. P. ROSS, and J. MAXWELL. Vol. LXIX. Demy 8vo. Pp. 413 + xxvi. Illustrated. 1936. London: John Murray. 21s. net.

Illustrations of Regional Anatomy. By E. B. JAMIESON, M.D., Senior Demonstrator and Lecturer, Anatomy Department, University of Edinburgh. F'cap 4to. Section VI, Upper Limb, 42 Plates, 7s. 6d. Section VII, Lower Limb, 52 Plates, 10s. 1936. Edinburgh: E. & S. Livingstone.

Disability Evaluation: Principles of Treatment of Compensable Injuries. By EARL D. MCBRIDE, B.S., M.D., F.A.C.S., Assistant Professor in Orthopedic Surgery, University of Oklahoma. Large 8vo. Pp. 623 + xvi, with 374 illustrations. 1936. London and Philadelphia: J. B. Lippincott Co. 38s. net.

Textbook of General Surgery. By WARREN H. COLE, M.D., F.A.C.S., Professor of Surgery, University of Illinois College of Medicine, and ROBERT ELMAN, M.D., Associate Professor of Surgery, Washington University School of Medicine, St. Louis. Large 8vo. Pp. 1031 + xvi, with 559 illustrations. 1936. London: D. Appleton-Century Co. Inc. 40s. net.

Modern Urology in Original Contributions by American Authors. Edited by HUGH CABOT, M.D., LL.D., C.M.G., F.A.C.S., Professor of Surgery, The Mayo Foundation, Graduate School of the University of Minnesota; etc. Third edition, thoroughly revised. Royal 8vo. In two volumes. Vol. I, pp. 951, with 546 illustrations and 12 plates; Vol. II, pp. 862, with 374 illustrations and 9 plates. 1936. London: Henry Kimpton. 90s. net.

Essentials of Oral Surgery. By Professor V. P. BLAIR, A.M., M.D., F.A.C.S., Washington University, St. Louis, and Professor R. H. IVY, M.D., D.D.S., F.A.C.S., University of Pennsylvania, Philadelphia, with the collaboration of Associate Professor J. B. BROWN, M.D., F.A.C.S., Washington University, St. Louis. Second edition. 9¼ + 6 in. Pp. 606, with 445 illustrations. 1936. London: Henry Kimpton. 30s. net.

The Digestive Tract: A Radiological Study of its Anatomy, Physiology, and Pathology. By ALFRED E. BARCLAY, O.B.E., M.A., M.D., D.M.R. & E., Honorary Radiologist to the Nuffield Institute for Medical Research, Oxford. Second edition. 7¼ × 9¾ in. Pp. 427 + xxxvi, with 296 illustrations, including 23 Plates. 1936. London: Cambridge University Press. 36s. net.

Urological Roentgenology. By MILEY B. WESSON, M.D., Ex-President, American Urological Association, and HOWARD E. RUGGLES, M.D., Roentgenologist to University of California Hospital, St. Luke's Hospital, and Clinical Professor of Roentgenology, University of California Medical School. 6¼ × 9¼ in. Pp. 269, with 227 illustrations. 1936. London: Henry Kimpton. 22s. 6d. net.

Medizinische Praxis. Band XXI. Anleitung zur Schmerzbetäubung. By Prof. Dr. FRITZ F. HÄRTEL, Direktor der chirurgischen Abteilung des Oskar Ziethen-Krankenhauses Berlin-Lichtenberg. Large 8vo. Pp. 106 + x, with 17 illustrations. 1936. Dresden and Leipzig: Theodor Steinkopff. Paper covers, RM. 10; bound, RM. 11.50.

Pathologie der Funktionen und Regulationen. By Dr. L. LICHTWITZ, Head of the Medical Department of the Montefiore Hospital, New York; Clinical Professor of Medicine, Columbia University. Large royal 8vo. Pp. 332 + viii, with 77 illustrations, mostly plates, and 61 graphs. Leiden: A. W. Sijthoff's Uitgeversmaatschappij N.V. Paper covers, Fl. 13.25; Bound, Fl. 14.50.

The Operations of Surgery. By R. P. ROWLANDS, M.S. (Lond.), F.R.C.S. (Eng.), Late Surgeon to Guy's Hospital, etc.; and PHILIP TURNER, B.Sc., M.S. (Lond.), F.R.C.S. (Eng.), Consulting Surgeon to Guy's Hospital, etc. Eighth edition. Vol. II: The Abdomen. Royal 8vo. Pp. 998 + ix, with 514 illustrations (4 in colour). 1937. London: J. & A. Churchill Ltd. 36s. net.

Tuberculosis, Cancer and Zinc. An Hypothesis. By DOUGLAS BARRON CRUICKSHANK, L.R.C.P. and S., L.D.S. (Edin.), D.P.H. (Camb.), Member of Research Staff, Papworth Village Settlement. With an Introduction by SIR PENDRILL VARRIER-JONES, M.A. (Cantab.), F.R.C.P. (Lond). Demy 8vo. Pp. 75 + xvi. 1936. London: Medical Publications Ltd. 7s. 6d.

Kidney Pain: Its Causation and Treatment. By J. LEON JONA, D.Sc., M.D., M.S., F.R.A.C.S., Hon. Assistant Gynæcological Surgeon, Women's Hospital, Melbourne. Large post 8vo. Pp. 94 + viii, with 61 illustrations. 1937. London: J. & A. Churchill Ltd. 7s. 6d.